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OF
OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

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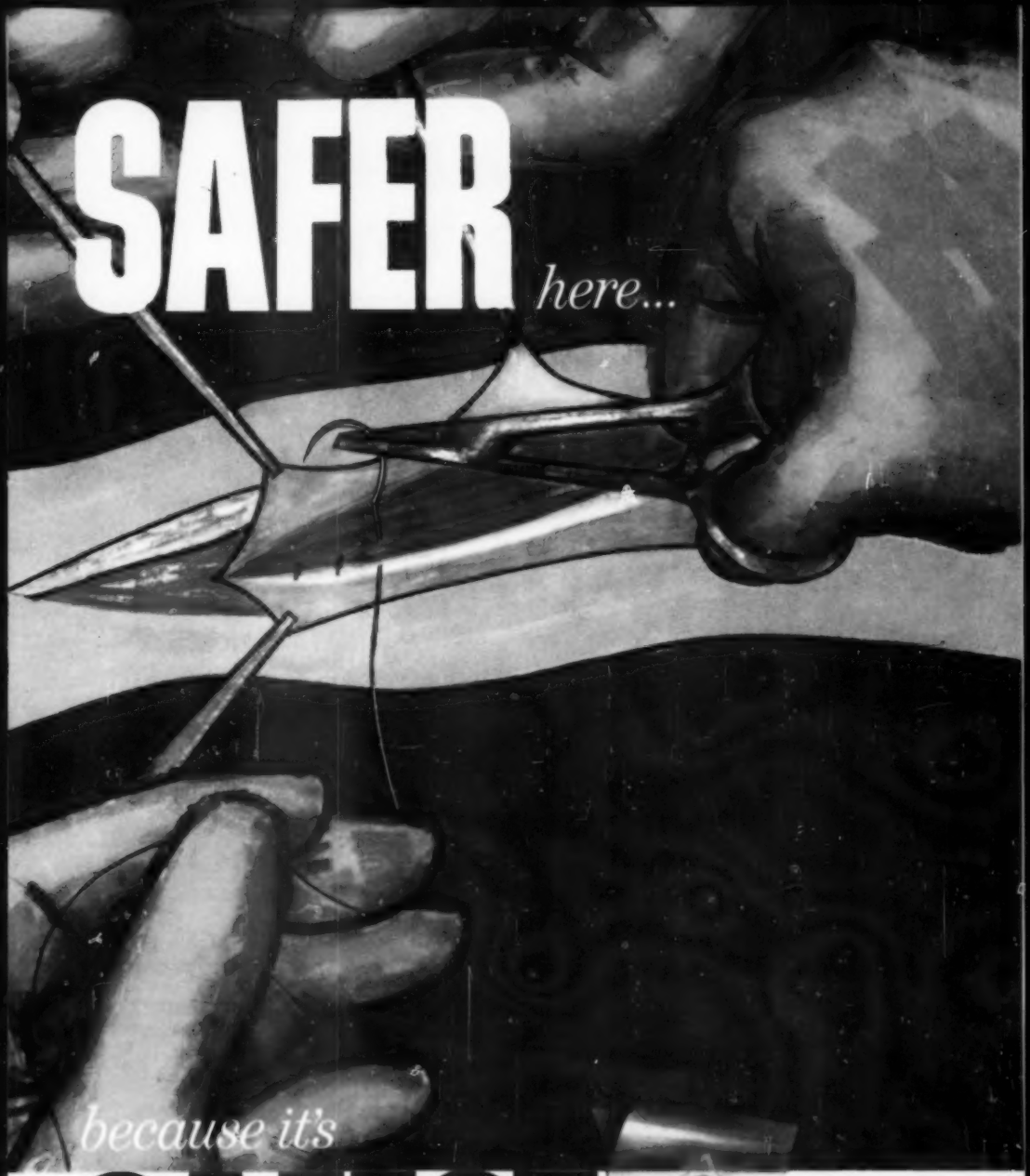

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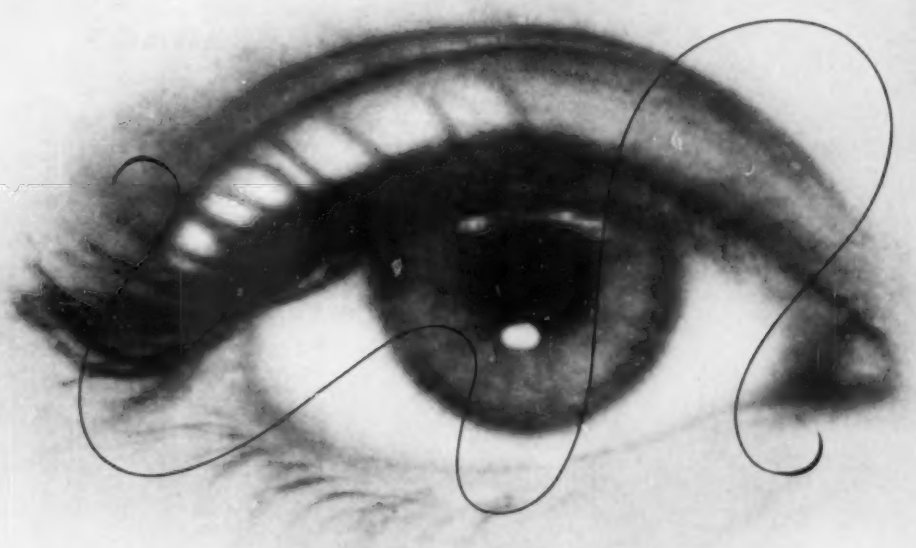

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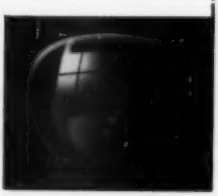
Panoptik  **Fused Lenticular Cataract**

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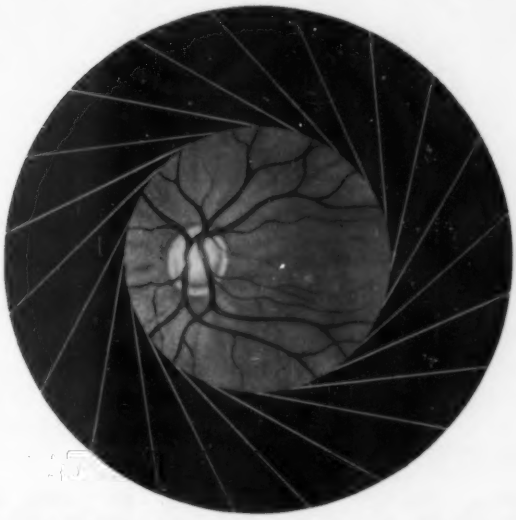

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1. Priestly, J. S.; Medina, M. M., and Phillips, C. C. To be published. 2. Ahlquist, R. P. in Drill, V. A.: Pharmacology in Medicine, McGraw-Hill Book Company, Inc. New York, 1954, p. 18-26.

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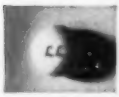
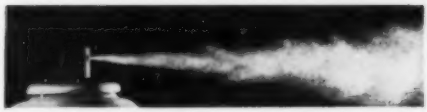
every  antibiotic 

need  ...

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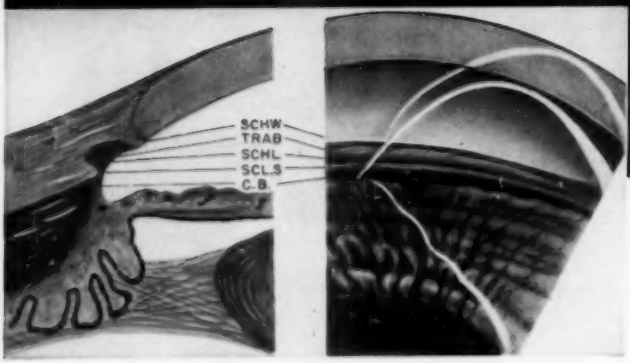

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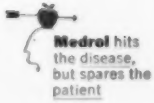

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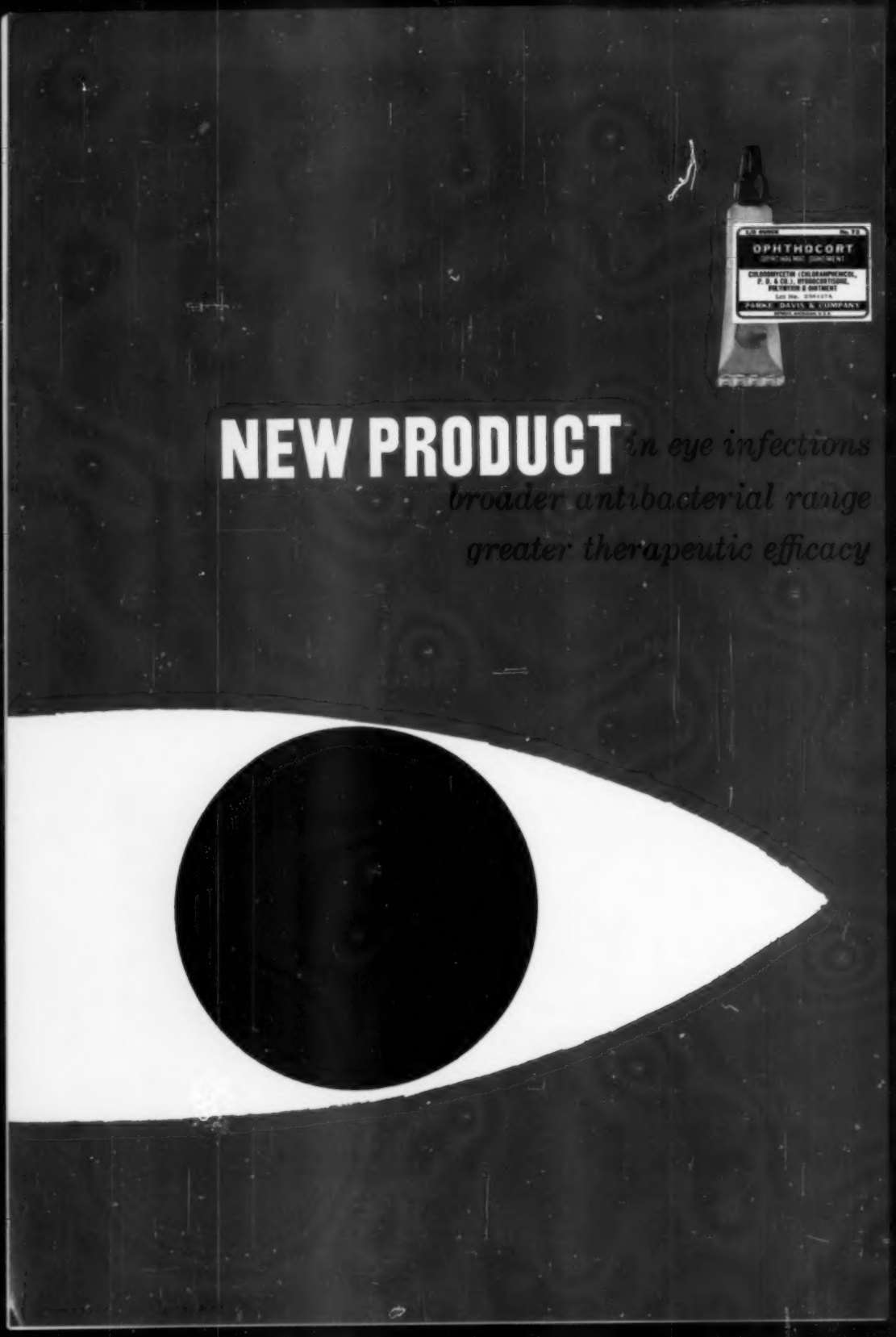
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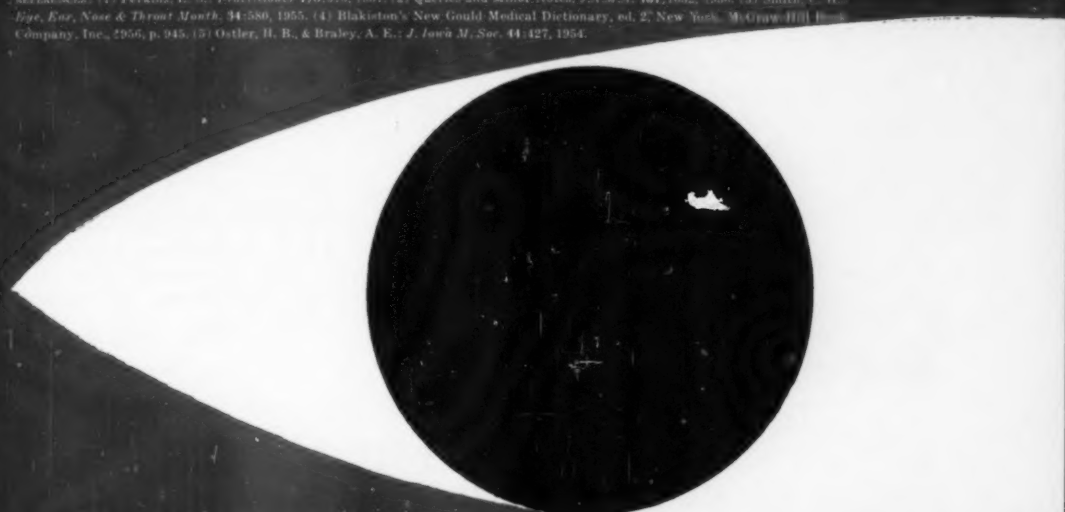
REFERENCES: (1) Perkins, E. S.: *Practitioner* 178:575, 1957. (2) Queries and Minor Notes, *J.A.M.A.* 161:1032, 1956. (3) Smith, C. H.: *Eye, Ear, Nose & Throat Month.* 34:580, 1955. (4) Blakiston's New Gould Medical Dictionary, ed. 2, New York, McGraw-Hill Book Company, Inc., 1956, p. 945. (5) Ostler, H. B., & Braley, A. E.: *J. Iowa M. Soc.* 44:427, 1954.



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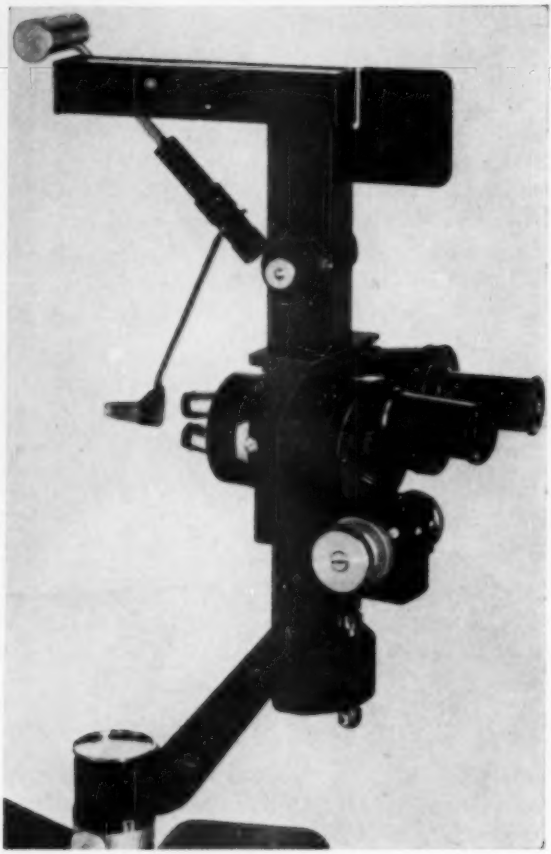

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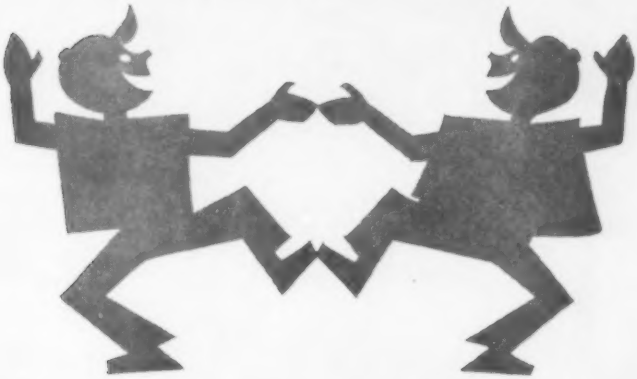
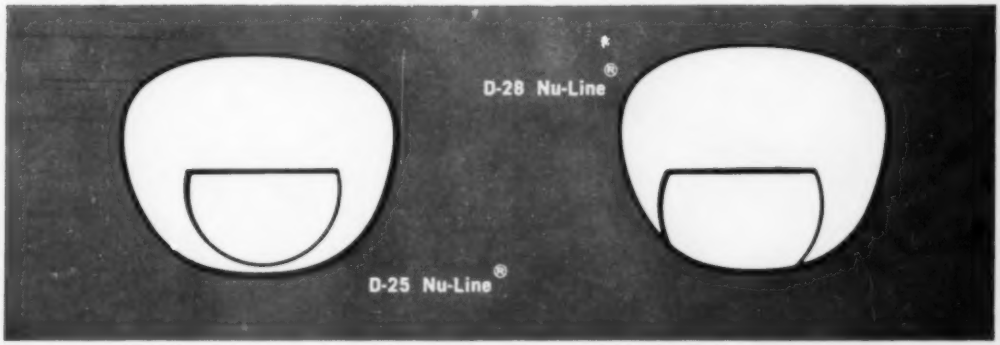


only the segment width changes



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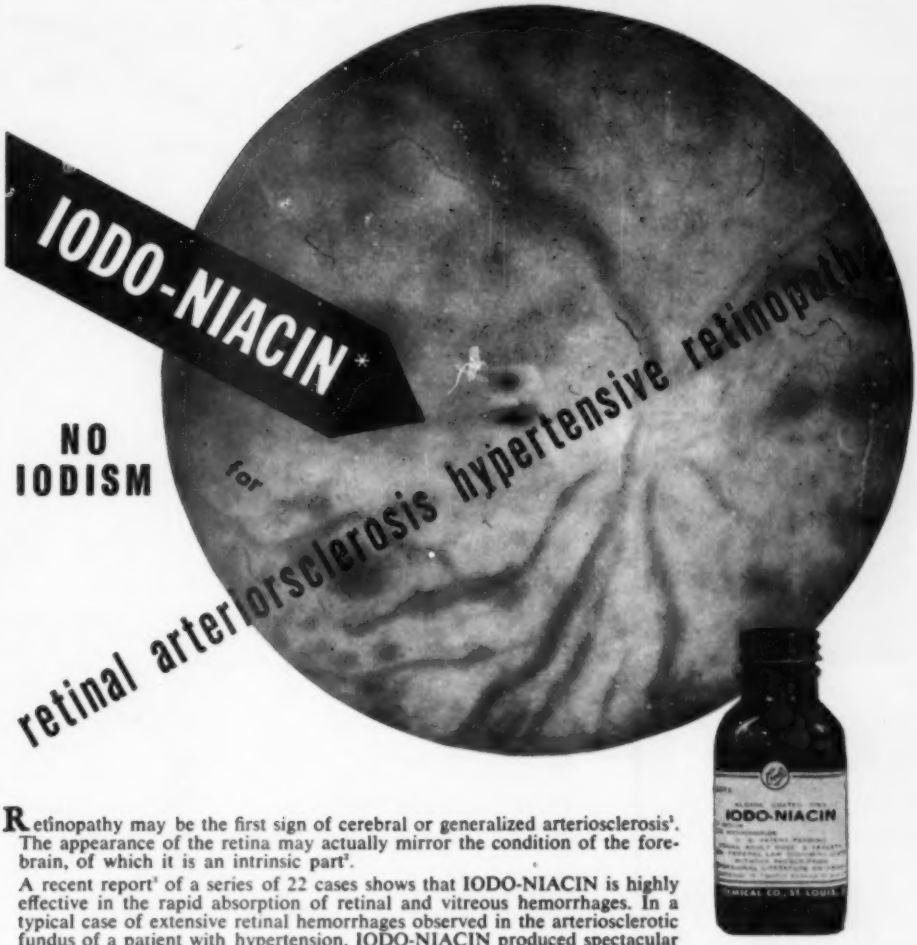

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1. *Cecil's Textbook of Medicine*, 7th ed., 1947, p. 1287.
2. *Ibid.*, p. 1598.
3. *Am. J. Ophth.* 42:771, 1956.
4. *Am. J. Digest. Dis.* 22:5, 1955.
5. *Med. Times* 84:741, 1956.

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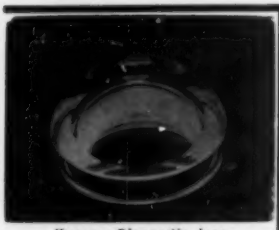

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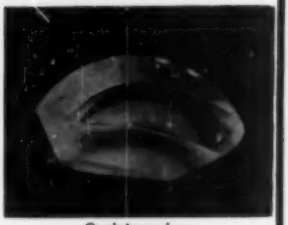

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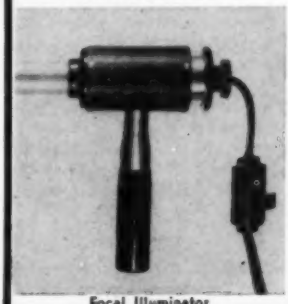

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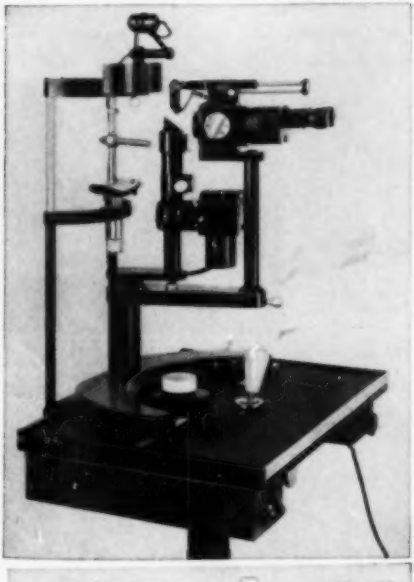

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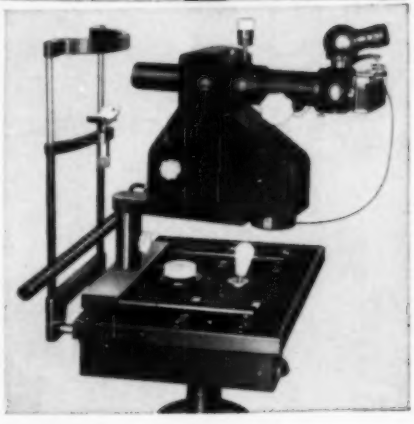

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Volume I

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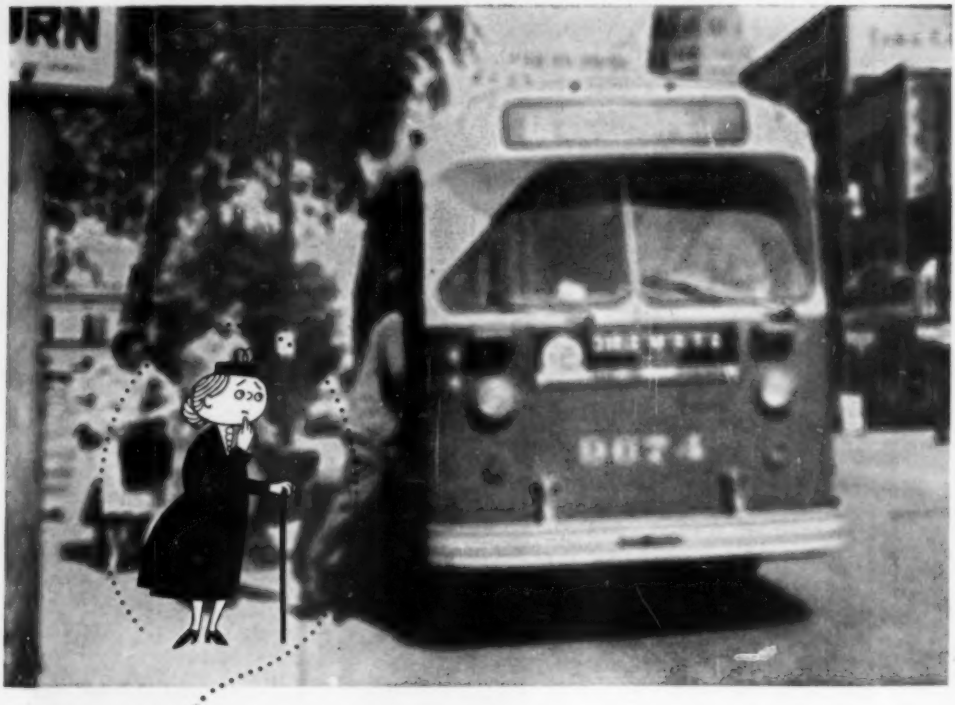

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THE IMPORTANCE OF CORRECT VERTICAL CENTERS

An interesting case occurred a few days ago which illustrates how two small errors can cause a lot of trouble.

We made two pairs of glasses for a railroad executive who does a great deal of close work. His prescription reads as follows—right: $+2.00\ +1.00 \times 180$; left: $+2.50\ +1.00 \times 180$.

He returned after getting his new prescription filled and complained that while one pair of glasses was very comfortable, the other pair could be worn for only a short time, after which they were most uncomfortable.

We checked horizontal and vertical centers, power and axis on both pairs. The power of the lenses and the axis of the cylinders were in strict accord with the prescription and identical. We found, however, that the distance center of the left lens in the pair causing trouble was one millimeter higher than the distance center of the right lens, whereas the distance centers in the comfortable pair were exactly the same height.

A difference of one millimeter in centers in a lens strength of $+2.00\ +1.00 \times 180$ would produce .3 degree of prism, base up, in that eye—enough to cause some trouble for a very sensitive pair of eyes. On closer examination of the prescription, we found that the left eye having a half diopter more power in the vertical meridian than the right eye would exercise a vertical imbalance at a reading point eight millimeters down from the distance center of .4 degree of prism, base up, in that eye. This imbalance, added to the .3 degree of prism caused by the distance center being one millimeter higher made a .7 degree of prism, base up, in the left eye, which was enough to cause the trouble.

The left lens was reground, the distance center moved downward one millimeter and the patient is now comfortable with either pair of glasses.

To some this small error in centering may seem insignificant, but in this particular case it represented the difference between a satisfied patient and a dissatisfied one.

"IF IT'S A LENS PROBLEM, LET'S LOOK AT IT TOGETHER"

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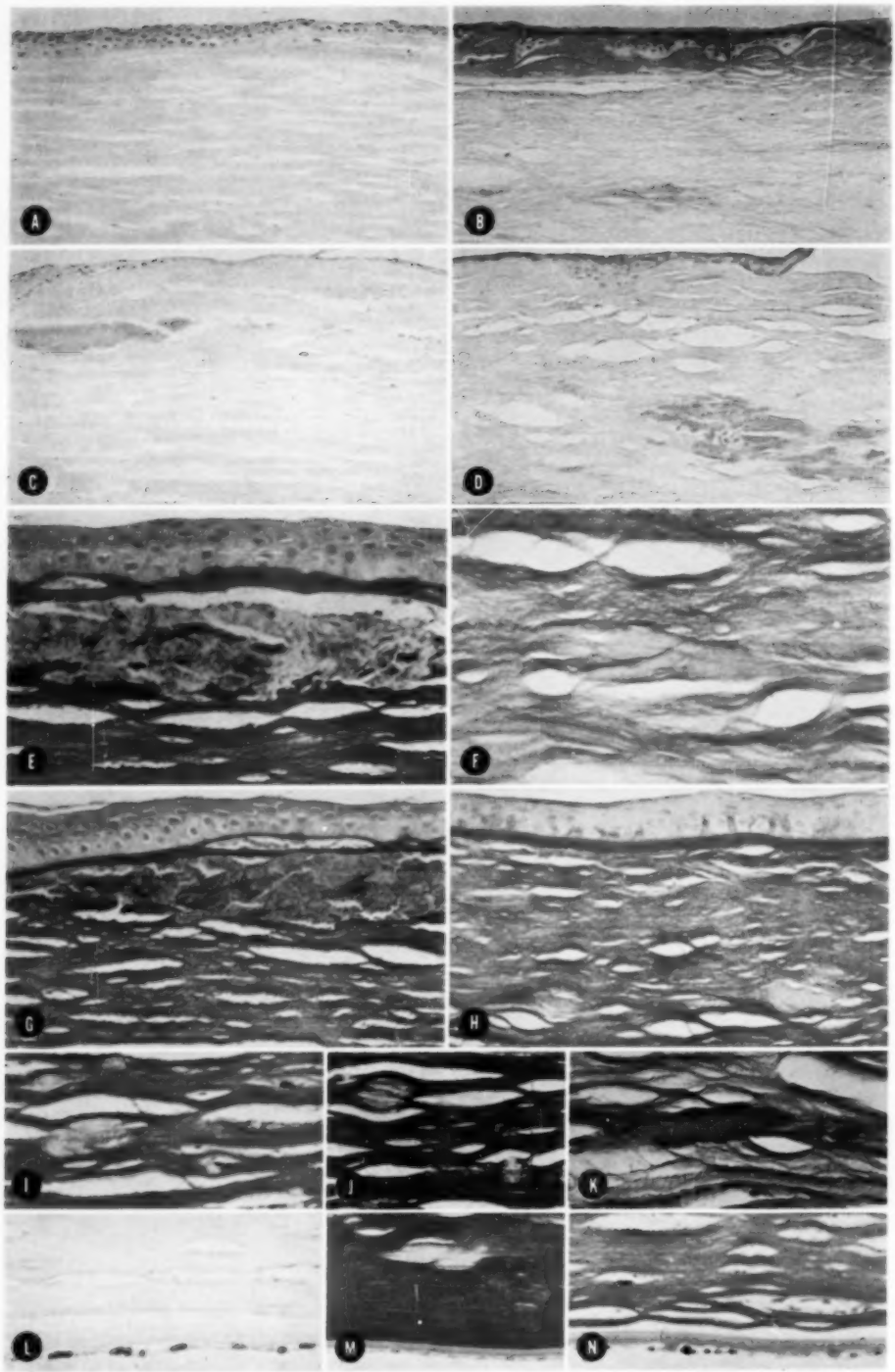
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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 47 JANUARY, 1959 NUMBER 1, PART I

MACULAR DYSTROPHY OF THE CORNEA (GROENOUW TYPE II)*

CLINICOPATHOLOGIC REPORT OF TWO CASES WITH COMMENTS CONCERNING ITS DIFFERENTIAL DIAGNOSIS FROM LATTICE DYSTROPHY (BIBER-HAAB-DIMMER)

SAM T. JONES, M.D., AND LORENZ E. ZIMMERMAN, M.D.
Washington, D.C.

In 1890 Groenouw[1] reported two cases of nodular corneal dystrophy; in the same year Biber,[2] a student of Haab, described the corneal lesions of three patients with lattice dystrophy. Since then, numerous instances of hereditary corneal dystrophy have been reported. In 1938 Bücklers[3] published his studies of the hereditary corneal dystrophies which occurred in 12 family trees, and showed that there were two types of Groenouw's nodular dystrophy, a granular form which is inherited as a Mendelian dominant and a macular variety which has a recessive hereditary pattern. Bücklers found that lattice dystrophy is separate and distinct from the other two forms. In each of the 12 pedigrees that he studied, Bücklers was able to find only one type of dystrophy per family.

* From the Registry of Ophthalmic Pathology, Armed Forces Institute of Pathology. Aided by a grant from The Ophthalmological Foundation, Inc., New York.

←

Color plate (Jones and Zimmerman). Macular dystrophy of the cornea. In the first four rows, typical lesions of macular dystrophy, on the left, are compared with those of lattice dystrophy on the right.

(A) Macular dystrophy (AFIP Acc. 840032). There is mucoid degeneration of corneal stromal fibers, most marked superficially just below Bowman's membrane. Early focal lesions of much smaller size are observed in deeper stromal fibers. A nonspecific plaque of hyalin superficial to Bowman's membrane lifts up the corneal epithelium in the upper right hand corner of the field. (Hematoxylin-eosin, ×115.)

(B) Lattice dystrophy (AFIP Acc. 220210). Several focal areas of hyaline degeneration of corneal stromal fibers are present in the lower half of the field. A much more extensive, but diagnostically less significant, deposit of hyalin is present between the irregularly atrophic epithelium and the partially destroyed Bowman's membrane. (Hematoxylin-eosin, ×115.)

(C) Macular dystrophy (AFIP Acc. 840032). An extensive zone of mucoid degeneration of stromal fibers is present just below the irregularly thickened Bowman's membrane. In the upper half of the field are seen varying degrees of conversion of the pink collagen fibers into a nonfibrillar substance which gives the typical blue-staining reaction characteristic of acid mucopolysaccharide. In the lower half, the collagen fibers remain intact and the mucoid deposits are confined to the interlamellar spaces, suggesting that they are in corneal corpuscles. Some mucoid material also appears incorporated within the subepithelial plaque of hyalin. (Alcian blue and nuclear fast red after hyaluronidase, ×130.)

(D) Lattice dystrophy (AFIP Acc. 220210). Those stromal fibers affected by the hyaline degeneration reveal no mucoid accumulations but they are intensely stained by the nuclear fast red. The nonspecific subepithelial deposits of hyalin are less intensely stained by the nuclear fast red. (Alcian blue and nuclear fast red after hyaluronidase, ×130.)

(E) Macular dystrophy (AFIP Acc. 840032). A large focal accumulation of material which gives the blue-staining reaction characteristic of acid mucopolysaccharide fills the defect formed by the disintegration of corneal collagen just below Bowman's membrane. Smaller accumulations of mucoid material are also seen between Bowman's membrane and the thickened basement membrane of the epithelium (upper left corner) and within the interlamellar corpuscles (lower half of field). (Rinehart-Abul-Haj method for acid mucopolysaccharides, ×305.)

(F) Lattice dystrophy (AFIP Acc. 220210). The affected stromal fibers stain yellow instead of red with the picrofuchsin counterstain. No accumulation of acid mucopolysaccharide is evident. (Rinehart-Abul-Haj method, ×305.)

(G) Macular dystrophy (AFIP Acc. 840032). In the superficial stroma collagen bundles are seen in varying stages of disintegration. This has resulted in an accumulation of finely dispersed nonfibrillar lavender material, some of which has dissected its way between Bowman's membrane and the basement membrane of the epithelium. (Weigert's resorcin-fuchsin stain, ×220.)

(H) Lattice dystrophy (AFIP Acc. 847258). The focally affected collagen fibers stain yellow with Weigert's resorcin-fuchsin stain. (×220.)

(I) Macular dystrophy (AFIP Acc. 840604). Small "early" lesions in the deep corneal fibers appear as swollen discrete foci which instead of staining red with the van Gieson counterstain appear pale blue. (Rinehart-Abul-Haj method, ×305.)

(J) Macular dystrophy (AFIP Acc. 840604). Similar lesions to those illustrated in (I). The affected corneal fibers appear as swollen poorly stained foci within the collagen bundles. (Masson's trichrome stain, ×265.)

(K) Lattice dystrophy (AFIP Acc. 220210). The focal areas of stromal involvement are stained bright red with Masson's trichrome. (×305.)

(L) Macular dystrophy (AFIP Acc. 840032). The corneal endothelium contains deposits which give a staining reaction characteristic of acid mucopolysaccharide. (Alcian blue and nuclear fast red after hyaluronidase, ×350.)

(M) Lattice dystrophy (AFIP Acc. 220210). A deep stromal lesion, stained yellow with picrofuchsin, is observed in the center of the field. No deposits of acid mucopolysaccharide are seen in the corneal corpuscles or in the endothelial cells. (Rinehart-Abul-Haj method, ×260.)

(N) Macular dystrophy (AFIP Acc. 840032). The endothelium and interlamellar corpuscles are filled with blue-staining globules believed to be acid mucopolysaccharide. Wartlike excrescences of Descemet's membrane are also present. (Rinehart-Abul-Haj method, ×260.)

TABLE 1

DISTINCTIVE CLINICAL CHARACTERISTICS OF THE THREE CLASSICAL HEREDOFAMILIAL CORNEAL DYSTROPHIES*

	Granular (Groenouw I) (Bücklers I)	Macular (Groenouw II) (Bücklers II)	Lattice (Biber-Haab-Dimmer) (Bücklers III)
Character of Opacities	Grayish opaque granules Sharp borders	Grayish opaque spots Borders not sharply delimited	Grayish lines like cotton threads, translucent by retroillumination; also rounded dots Distinct borders
Cornea Intervening Between Opacities	Clear	Diffusely cloudy	Relatively clear
Distribution of Opacities	In a disc-shaped area in central (axial) region of cornea only. The peripheral portion of the cornea in a zone 1–3 mm. wide is always free of granules	Scattered over entire cornea, more dense in axial region. Some spots extend to the limbus. (The central opacities may be more superficial, the peripheral opacities, deeper.)	Rounded dots scattered everywhere. Threadlike lines mainly limited to a zone between the center of the cornea and the periphery. (Usually do not extend to limbus.)
Vision	Good until middle or old age. Considerably reduced only by the 50th to 60th year of life.† Of 91 patients reported by Bücklers, only 2 had such poor vision that they were unable to do their work	Affected early in life, usually considerably reduced by age 30 to 40. Reduced to finger-counting at a few meters (or less) by age 40 to 50.	Reduced early in life (20–30 years). May be reduced to 20/200 by age 40 to 50.
Hereditary Transmission	Dominant	Recessive	Dominant

* Table modified after Bücklers[5] and Mutch.[6]

† Franceschetti and Kiewe[7] reported an unusual family with granular dystrophy in which the vision was definitely diminished around the age of 20 to 30 years.

He found no evidence of transitional forms between the three dystrophies. Between families considerable variation existed in the appearance of each type of dystrophy, but there was remarkable constancy of the clinical appearance of the diseased corneas within the same family.†[4]

The distinctive clinical characteristics of the three classic heredofamilial corneal dystrophies are summarized in Table 1. It should be noted that grayish opacities appear in the corneal stroma in both granular and macular dystrophy, but that the corneal disease is more severe in macular dystrophy, which is inherited as a recessive trait. This conforms to one of the general patterns of hereditary diseases: those with recessive inheritance tend to produce more severe clinical symptoms.[8]

Although their clinical and genetic features have been extensively reported, relatively few papers have been published reporting the pathologic anatomy of the granular, macular, and lattice dystrophies. In 1902 Ernst Fuchs[9] reported performance of the first corneal transplantation in a case of Groenouw's nodular dystrophy (probably macular dystrophy), and included his histologic observations on sections of the excised corneal button. Now, with improvement in the surgical technique of corneal transplanta-

† Granular dystrophy in children is apt to be confused with lattice dystrophy because the minute granules found in children are frequently arranged in radiating lines. As the patient reaches the second and third decades of life, the corneal opacities in granular dystrophy "come together to form tiny circles, discs, ovals, streaks, bands, arches, half-moons (crescents)." . . .[4]

tion, a number of patients with hereditary corneal dystrophies are being treated surgically, and the corneal buttons removed are becoming available for histopathologic study.‡ The corneal buttons from the following two cases of macular dystrophy became available for microscopic examination as a result of corneal transplantation.§

CASE 1. (C. A., AFIP Acc. 840032)

This 55-year-old man had no eye symptoms when he immigrated to the United States from southern Italy in 1919, at the age of 17 years; however, a doctor at the immigration station told the patient that he had "keratita." Four years later while shooting a gun, he noted that his vision was not as good as it had been. After that, his vision became progressively worse until he was 48 years of age, at which time he first came to the Illinois Eye and Ear Infirmary. He stated that he had been unable to read since the age of 43 years.

The patient's father died at the age of 77 years, his mother at the age of 67. Both had good vision until the time of their deaths. The patient had 11 siblings: one brother died at age 34 years, another at 70, and another at 72; one sister died at the age of 70 years; the remaining siblings (four males, three females) ranged in age from 53 to 63 years. None of the siblings has had any visual difficulty except for a brother, aged 63 years, who lives in Italy. This brother has had poor vision in both eyes since the age of seven years, and is now said to be virtually blind. The patient knows of no other near or distant relative who has had any visual impairment that was not correctable with glasses. The patient has one child, a daughter, aged 23 years, whose corneas are perfectly clear on slitlamp examination. There is no family history of consanguineous marriages.

Examination in December, 1950, when the patient was 48 years of age, revealed the vision to be finger-counting at four feet in the right and finger-counting at two feet in the left eye. There was good light projection. There was no conjunctival congestion. Both corneas presented a similar appearance (fig. 1). The epithelium was uneven, being elevated over small rounded confluent opaque spots located superficially. The cornea intervening between these dense opacities was not clear, but was diffusely cloudy. The dense superficial opacities were more marked in the axial region of the cornea; peripherally, smaller whitish opacities were located in the region of Descemet's membrane. These small deep opacities extended to the limbus. Several Hudson-Stähli lines were present. The corneal sensitivity was markedly diminished.

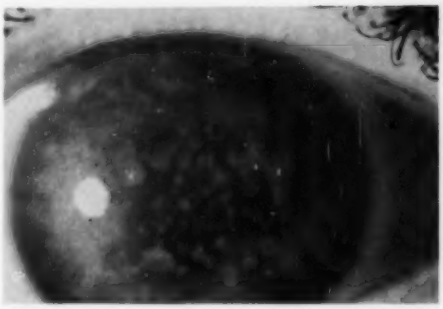

Fig. 1 (Jones and Zimmerman). *Case 1.* Preoperative appearance of right cornea.

The clinical diagnosis was macular corneal dystrophy, Groenouw Type II.

On February 7, 1951, a five-mm. penetrating corneal transplantation was performed on the left eye. Overlying sutures were used. The postoperative course was complicated by forward bulging of the graft persisting for several weeks. Two years following keratoplasty, the vision in the left eye with a contact lens was 20/30. The patient now wears a spectacle lens with which his vision in the left eye is 20/30.

In January, 1957, the patient was examined for surgery on the right eye. The vision in the right eye was still finger-counting at four feet.

On February 2, 1957, a six-mm. penetrating corneal transplantation was performed on the right eye, 11 direct sutures being used to fixate the graft to the host cornea. At the time of the operation, the patient's cornea was noted to be about two thirds the average normal thickness. The donor cornea was slightly edematous and was somewhat greater than average normal thickness. Postoperatively the patient developed a local sensitivity reaction to atropine. On the 13th postoperative day, all the sutures except those at 3-, 6-, 9-, and 12-o'clock positions were removed. After suture removal there was separation of the wound edges, especially from the 12- to 3-o'clock position, and the graft appeared to be tilted. The edge of the graft projected above the surface of the host cornea from the 12:30- to the 2:30-o'clock position.

On the 19th postoperative day, a membrane was noted on the endothelial surface of the graft, extending inward like a tongue from the 9:30- to the 11:30-o'clock position. This membrane extended inward about one mm. when first seen, and over the next few months extended inward another 0.5 mm. The four remaining sutures were removed about six weeks postoperatively. The elevated edge of the graft gradually became less prominent and the membrane on the posterior surface of the graft

‡ As other workers have pointed out, the corneal buttons obtained as a result of keratoplasty usually show the later stages of the disease process and do not permit histologic examination of the peripheral cornea.

§ Since the vision of patients with macular dystrophy is severely affected at a relatively early period in life, they are particularly likely to have corneal surgery performed.

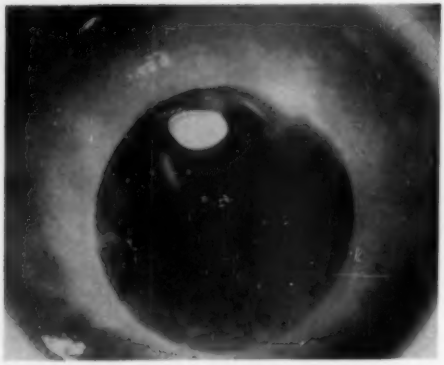

Fig. 2. (Jones and Zimmerman). *Case 1.* Appearance of right cornea six and one-half months after keratoplasty.

failed to extend any farther centrally. On June 26, 1957, the vision in the right eye was 20/30 with a spectacle lens (fig. 2, taken August 17, 1957).

Pathologic study, Case 1

The excised button of cornea obtained in 1957 was embedded and sectioned in paraffin. These sections revealed the epithelium to be intact but of variable thickness. In places where the epithelium lay directly upon Bowman's membrane, it showed only mild edema and varied but slightly in thickness (color plate A). In several places, however, changes in the subepithelial tissues were associated with pronounced alterations in the overlying epithelium (fig. 3 and color plate C). Varying degrees of degeneration and atrophy of the epithelium were observed in such areas, the epithelium being reduced to a single layer of extremely flattened cells over the summit of some of the larger subepithelial plaques.

The subepithelial alterations included several different processes. The most subtle change was an increased prominence of the basement membrane in focal areas (fig. 4 and color plate—E and G). In such foci the latter was not only thickened but it took on different staining characteristics which led to its being readily differentiated from Bowman's membrane, not only with the reticulum stain and with the periodic acid-Schiff (PAS) method but also to a lesser degree with such other stains as the Verhoeff-van Gieson, Weigert's resorcin-fuchsin, and the May-Grünwald-Giemsa. In places the thickening of the basement membrane was associated with an accumulation of hyalin, and there were minute deposits of a mucoid material which with hematoxylin-eosin stained a very faint gray. The staining characteristics of this mucoid material will be described in greater detail later. Bowman's membrane, for the most part, was intact and perhaps slightly thickened. In the areas where the largest accumulations of hyalin and mucoid material were present between the epithelium and Bowman's membrane, the latter tended to be more irregularly thickened. In these areas it also appeared to be reduplicated, frayed, and obscured by the associated deposits of hyalin.

The most distinctive alterations were found in the superficial portions of the substantia propria. There were many places in which the corneal lamellae displayed a most peculiar form of disintegration, best characterized as mucoid degeneration. In the affected areas the stromal fibers became markedly frayed and in the larger lesions the fibrillar character of the stroma was completely destroyed. In these most severely affected areas the collagen bundles were replaced by amorphous masses of material which with hematoxylin and eosin appeared very pale staining, faintly grayish blue (fig. 3 and color plate A). Where the collagen bundles were not completely disintegrated, extremely fine pink fibrils could still be discerned within the masses of grayish blue mucoid material. Typically there was a pronounced loss or complete absence of the normal birefringence of the corneal collagen in these lesions (fig. 3-B). For the most part, these were completely acellular lesions, even the corneal corpuscles having disappeared from the affected fibers (fig. 5). In some of these lesions, however, the corneal corpuscles remained but appeared extremely degenerated. Their nuclei were irregularly pyknotic

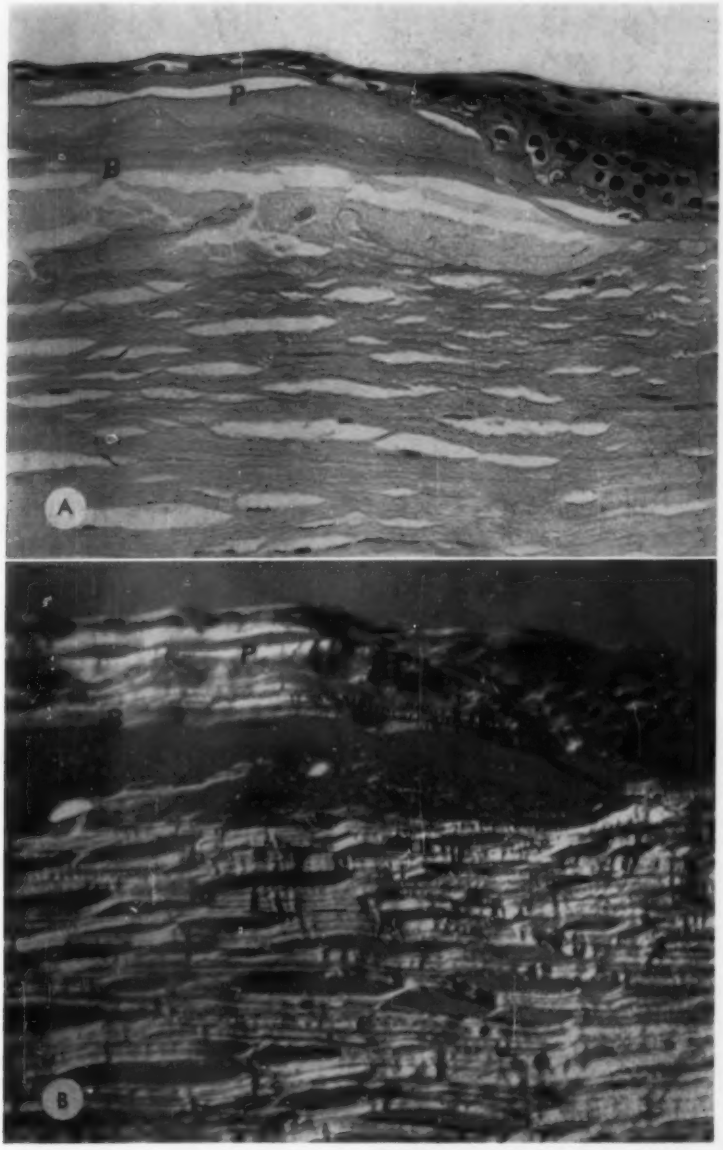

Fig. 3 (Jones and Zimmerman). AFIP Acc. 840032. *Macular dystrophy.* The markedly degenerated epithelium is reduced to a single layer of cells over a moundlike hyaline plaque (P) which rests upon Bowman's membrane (B). Beneath Bowman's membrane there is a large area of mucoid degeneration of the stromal lamellae. The degree of disintegration of the collagenous fibers is better demonstrated by examination with polarized light. The absence of birefringent fibers in the lesion beneath Bowman's membrane is in distinct contrast with the doubly refractile material present in the deeper stromal layers and in the plaque above Bowman's membrane. (A) Hematoxylin-eosin, ordinary illumination, ×305. (B) Hematoxylin-eosin, polarized light, ×305.

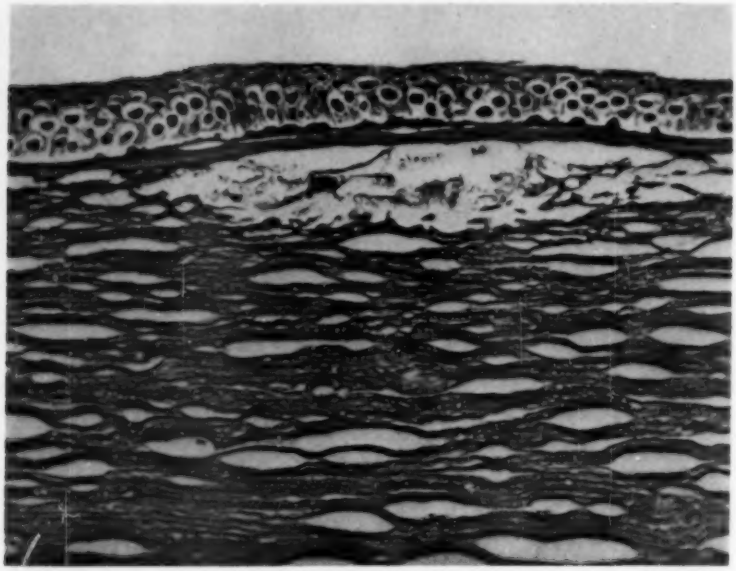

Fig. 4 (Jones and Zimmerman). AFIP Acc. 840032. *Macular dystrophy.* The basement membrane is thickened and separated from Bowman's membrane in the center of the field. The severity of stromal fiber disintegration within the specific lesion beneath Bowman's membrane is beautifully demonstrated by the Wilder stain for reticulum. (×305.)

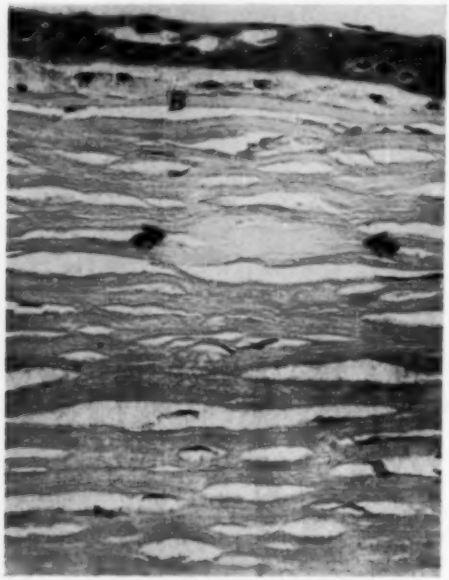

Fig. 5 (Jones and Zimmerman). AFIP Acc. 840032. *Macular dystrophy.* A focal acellular area of early mucoid degeneration of the corneal lamellae is indicated by arrows. Large mononuclear cells, presumably wandering histiocytes filled with acid mucopolysaccharide, are present between Bowman's membrane (B) and the epithelium. (Hematoxylin-eosin, ×305.)

and their cytoplasm greatly distended by faint grayish blue mucoid material similar in appearance to that which characterized the acellular areas of collagen disintegration.

The staining characteristics of the mucoid deposits already described in the superficial corneal stroma and in the plaques lying between the epithelium and Bowman's membrane were very distinctive. With the Rinehart-Abul-Haj[10] (color plate E) and Alcian blue[11] (color plate C) techniques for staining of acid mucopolysaccharide, they appeared faint blue and finely granular. With Weigert's resorcin-fuchsin (color plate G), they seemed more coarsely granular and stained grayish purple. The granules were intensely PAS-positive but they were virtually un-

stained by MacCallum's modification of the Gram stain. The connective tissue stains (color plate I and J) including Wilder's method for reticulum (figs. 4 and 6) revealed best the disintegration of the fibrillar architecture of the corneal lamellae in the areas of mucoid degeneration. The staining reaction of the mucoid deposits were not altered by pretreatment of the sections with hyaluronidase or with diastase.

Although the most extensive areas of mucoid disintegration of the corneal lamellae were observed in the outer third of the substantia propria, smaller foci believed to represent earlier stages of the same process were observed with decreasing frequency as successively deeper layers of the substantia were examined. In the middle layers many focal areas of swelling could be seen within the affected lamellae. Such foci were pale-staining with hematoxylin and eosin (fig. 5) and were characterized by a loss of the fibrillar architecture of the collagen bundles as seen with the connective tissue stains, especially Masson's trichrome (color plate J) and Wilder's reticulum (fig. 6). With the Rinehart-Abul-Haj stain for acid mucopolysaccharide, these lesions were characterized by focal accumulations of pale blue-staining material against the ordinarily red-staining background (van Gieson counterstain) of the corneal stroma (color plate I). Similarly positive reactions were obtained in the affected corneal fibers when alcian blue was used.

In addition to these focal changes occurring within the substance of the corneal lamellae, there was a widespread accumulation throughout the interlamellar spaces of granular material which gave the intensely positive staining reactions for acid mucopolysaccharide (color plate C, E, I, L, and N). It was frequently obvious (especially with the Alcian blue-nuclear fast red combination) that this material was within the cytoplasm of corneal corpuscles. In many places, however, it was not possible to prove conclusively that the material was entirely intracytoplasmic, for neither nuclei nor cytoplasm of corneal corpuscles were demonstrable in the planes of section. The distribution and general appearance of these interlamellar accumulations strongly suggested that all probably represented massive accumulations of acid mucopolysaccharide within the cytoplasm of the stromal cells. The staining characteristics of these interlamellar accumulations were the same as in the larger superficial areas of stromal disintegration.

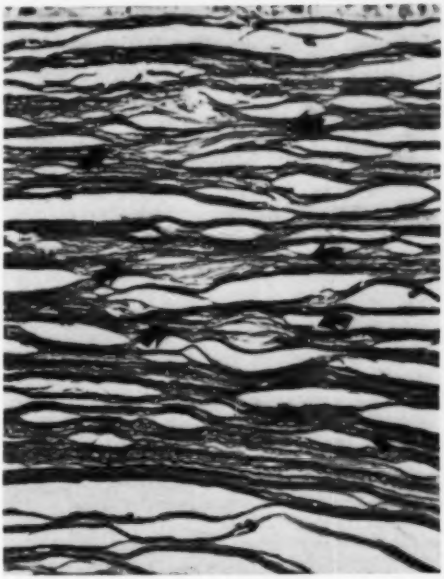

Fig. 6 (Jones and Zimmerman). AFIP Acc. 840032. *Macular dystrophy.* Foci of early disintegration of the fibrillar architecture of the corneal lamellae are revealed by the Wilder reticulum stain (arrows). (×305.)

There was diffuse thickening of Descemet's membrane, which, with several staining methods, particularly the PAS preparations, revealed a fine laminated pattern. In addition to the diffuse thickening, Descemet's membrane exhibited many rectangular and anvil-shaped wartlike excrescences protruding through the endothelium toward the anterior chamber (color plate N). The cytoplasm of the endothelial cells frequently was engorged with granular material identical

in its staining reaction to the mucoid deposits described within the corneal stroma (color plate L and N).

CASE 2 (O. H., AFIP Acc. 840604)

This 45-year-old man, a school administrator, began to wear glasses for astigmatism at the age of 12 or 13 years. At this age he was told that his eyes were "hazy or not clear." He entered college at the age of 19 years, and noted for the first time that even with new glasses he could not read the blackboard assignments from a distance. When he was 20 years of age, his right eye was injured by a fingernail scratch during a football game, and after this he could not see as well as previously with his right eye. While he was in his early twenties, he had difficulty seeing golf balls on the fairway. When he was 29 years of age, he was examined at the Mayo Clinic, where a diagnosis of Groenouw's dystrophy was made and where his vision was found to be 20/40 in the right eye and 20/50 in the left eye. During his early thirties he was rejected for military service on account of his eyes. At the age of 35 years he began to use a loupe to read books.

The patient's father, who died at the age of 64 years, never had visual difficulty and was able to work as an accountant until shortly before his death. The patient's mother, who died at the age of 56 years, had no difficulty seeing and could read books until the time of her death. The patient had six siblings: one brother died at the age of 33 years; the other siblings (three females, two males) ranged in age from 48 to 58 years. None of the siblings has had any visual difficulty except for a sister, aged 58 years, who had severe bilateral impairment of vision while she was in her thirties. This sister had bilateral corneal transplantations at the age of 51 and 52 years, with good results. The patient knows of no other relative with visual impairment that was not correctable with glasses. The patient has no children. There is no family history of consanguineous marriages. The family is of German origin.

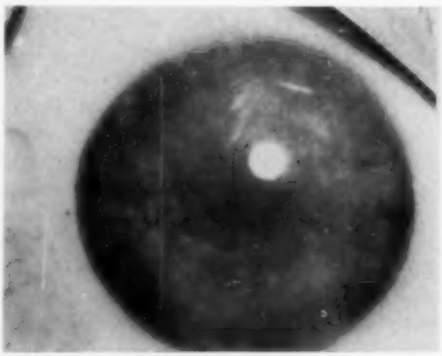

Fig. 7 (Jones and Zimmerman). *Case 2.* Appearance of patient's left cornea.

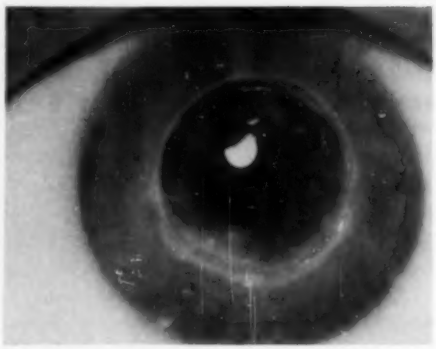

Fig. 8 (Jones and Zimmerman). *Case 2.* Appearance of right cornea eight months after keratoplasty.

Examination on June 10, 1957, revealed the vision in both eyes to be finger-counting at two feet. The two corneas had the same appearance (fig. 7). The epithelium was smooth. The stroma was filled with confluent opacities, which were very dense and superficial in the central portions of the cornea, and which were less dense and located more deeply in the peripheral portions of the cornea. The corneal stroma intervening between the opacities was not clear, but was diffusely cloudy. The corneal sensation was severely impaired.

The clinical diagnosis was macular corneal dystrophy, Groenouw Type II.

On June 25, 1957, a six-mm. penetrating corneal transplantation was performed on the right eye. The postoperative course was uneventful until after the removal of the sutures. Following suture removal, the graft bulged forward, and there was marked elevation of the graft edge inferiorly. On August 7, 1957, the vision in the right eye was 20/300 with pinhole, and the graft edges were beginning to fill in. A few posterior synechias were noted nasally.

On September 26, 1957, the vision in the right eye was 20/100 without glasses. With a spectacle lens, the patient could see 20/50 at a distance, and with the bifocal segment he could read five-point print. In September, 1957, the lower one fifth of the graft was still somewhat thickened but the graft became clear over the next few months and has remained transparent (fig. 8). Surgery on the left eye is planned for the near future.

Pathologic study, Case 2

The corneal button obtained from this patient's right eye was processed and sectioned in paraffin. The superficial changes observed in this cornea were much less pronounced than were those in Case 1. There was relatively little irregularity of the epithelium and no large subepithelial plaques were

present upon Bowman's membrane. In the central part of the button, Bowman's membrane appeared to be involved along with the superficial corneal lamellae in a process of mucoid disintegration essentially comparable to that seen in Case 1. The changes here, however, were much less extensive in the superficial areas and the resultant accumulations of mucoid material were considerably less massive. The focal changes observed in individual corneal bundles in the middle and deeper layers, however, appeared to be more numerous and more widely scattered than in Case 1. The essential character of these lesions, however, was identical. In this button the diffuse interlamellar accumulations presumed to represent deposits within the corneal corpuscles were comparable to those of Case 1, as were the accumulations within the cytoplasm of the endothelial cells.

With the various staining procedures, the morphology and tinctorial features of the mucoid material observed in the various locations appeared to be identical with those described for Case 1, and they will therefore not be described again.

There was diffuse thickening of Descemet's membrane though less marked than observed in Case 1. Occasional sessile wartlike protuberances were also observed but this feature was much less exaggerated than in Case 1.

Histopathology of Macular Dystrophy

Through the courtesy of several ophthalmic pathologists we have been able to secure corneal specimens from patients who have had a variety of corneal diseases. This material has included buttons from eight patients with macular dystrophy and from five patients with lattice dystrophy. As a result of our study of this material we arrived at the conclusion that the fundamental tissue alteration in these two diseases was very different, sufficiently so to permit sharp histopathologic differentiation. Consulting such standard references as Duke-Elder[12] and the volume on *Ophthalmic Pathology* by Friedenwald and co-workers,[13] we were impressed by the fact that it is not generally appreciated that such histopathologic differentiation is possible. Furthermore, we had to go back more than 50 years to Fuchs' work[9] to get a clear-cut description of the mucoid nature of the basic lesion of macular dystrophy.

In 1902 Ernst Fuchs[9] published his histopathologic observations made on sections of a corneal button trephined from a patient with macular dystrophy.‖ Fuchs noted that the superficial lamellae were pink with the van Gieson stain while the deeper lamellae were darker red. In some places the superficial lamellae were swollen and more homogeneous than normal, with loss of the normal lengthwise striping corresponding to the lamellar fibrils. Among these superficial layers there were certain places where the lamellae were separated or frayed out into fibrils which united and then separated again, leaving large interspaces between them. These places corresponded to the macroscopically visible large gray spots seen on the corneal surface. Between the superficial lamellae, as well as between the deeper lamellae, which appeared more normal, Fuchs found an amorphous substance which was gray in the hematoxylin-eosin preparation. With low magnification, the substance looked homogeneous; with higher magnification, it appeared finely granulated and seemed to be traversed by a very fine network of fibrils. Fuchs thought that in the living cornea the amorphous substance was a colloid, which was "curdled" by the fixation fluid, giving it the granulated appearance seen in the sections. These collections of amorphous material appeared to contain the remains of corneal corpuscles undergoing destruction. The amorphous substance was found to be most abundant where the changes in the superficial lamellae were most advanced, that is, in those places where

‖ The button was 0.25-mm. thick. It was obtained as a result of a four-mm. lamellar corneal transplantation.

the separation into fibers had produced nodules. In the larger nodules, therefore, the following sequence of layers was seen from above downward: (1) the thinned epithelium, (2) lamellae separated into fibers, (3) the amorphous mass, and (4) normal corneal lamellae.

In the parenchymal layers below the swollen lamellae, Fuchs found an abnormal metachromatic staining reaction with thionine; this led him to assume the presence within the lamellae of a mucoid substance which later became transformed into the amorphous substance between the lamellae. The amorphous material lying in the interlamellar spaces did not stain metachromatically with thionine.

In 1915 Fuchs[14] reported performing lamellar keratoplasties in two additional cases of macular dystrophy. In a communication beautifully illustrated by a colored plate, he demonstrated that the amorphous material was colored pale blue when a Giemsa stain was used. He also reported that in addition to the blue amorphous substance, there was a red-staining plaque between the epithelium and Bowman's membrane.

In 1950 Franceschetti and Babel[15] published a review of the pathologic anatomy of of the heredofamilial degenerations of the cornea. They reported that in macular dystrophy "the lesions are more diffuse than in the dominant forms and extend to all layers of the cornea." They observed a "hyaline degeneration, identical with that found in the dominant forms. This is mainly localized to the anterior layers of the stroma. . . . All the fixed cells of the cornea are abnormally swollen and filled with granules. . . . Most frequently with the ordinary stains (hematoxylin-eosin, May-Grünwald-Giemsa, Gram, van Gieson) the granules are invisible or only slightly perceptible. When stained with resorcin-fuchsin they appear dark violet-gray in color. . . . In the anterior layers, transformed by the hyalinosis the number and the size of the granules increase markedly and one can see enormous violet-grayish heaps." In 1945 Franceschetti and Babel[16] had reported finding large deposits of this granular material within the endothelial cells in macular dystrophy.

Wolter and Cutler,[17] in a recent article entitled "Granular dystrophy of the cornea," described their histopathologic observations on a corneal button removed from a 39-year-old woman. On the basis of the clinical data —vision affected at an early age, recessive hereditary pattern, and the clinical appearance and distribution of the corneal opacification—it seems to us that this patient had macular dystrophy, rather than granular dystrophy. These workers described their pathologic observations based on flat sections cut with the freezing microtome and stained by neuropathologic methods. Their histologic preparations showed that there was severe damage to the corneal corpuscles in the superficial stroma, while the cells became more and more normal toward the posterior stroma. It was their opinion that "the degeneration of the stromal cells of the cornea represents the primary pathologic defect and that the hyalinization of the corneal lamellae occurs secondary to the cellular changes." In the deeper layers, where only the stromal cells deviated from normal, Wolter and Cutler observed that: "Their cell bodies and processes appeared somewhat swollen, and the protoplasm contained vacuoles of different sizes."

Franceschetti and Babel,[15] who also employed silver impregnation, had previously described vacuoles in the corneal corpuscles in macular dystrophy. They reported that these vacuoles did not contain lipoidal material. From our own studies, we believe that the material in the cell bodies is an acid mucopolysaccharide.

In addition to the two cases reported in detail, we have had the opportunity to make histopathologic observations in six other cases of macular dystrophy in which corneal transplantations had been performed. As a result of our own studies, we have come to the conclusion that the essential alteration is

one of mucoid disintegration of the fibers within the substantia propria of the cornea. In addition to the focal areas of destruction within the collagenous bundles, there are widespread alterations in the corneal corpuscles and in the endothelial cells. The tremendous accumulations of granular material which give the typical staining reaction of acid mucopolysaccharide and which are found throughout all of the cellular elements of the corneal stroma might be considered indicative of the sites of the primary damage, as suggested by Franceschetti,[15] Wolter,[17] and their collaborators; but they could, in our opinion, be secondary manifestations. It seems to us that as a result of the mucoid disintegration of the stromal bundles, the accumulated mucoid material does not remain focalized to the areas of collagen disintegration, but rather these accumulated materials diffuse out into portions of the cornea where the substance is phagocytosed by the corneal corpuscles and by the endothelium. Likewise the minute deposits superficial to Bowman's membrane in Case 1 are interpreted as secondary rather than as primary manifestations of the basic process.

In keeping with the suggestion that the abundant deposits of mucoid material found within the endothelial cells represent a secondary feature of the basic process, we may then suggest that the alterations in Descemet's membrane represent a tertiary phenomenon. The diffuse and nodular thickening of Descemet's membrane, reminiscent of the changes observed in Fuchs' dystrophy of the cornea, might be the consequence of disturbed metabolism of the corneal endothelium incident to the tremendous accumulation of abnormal materials within its cytoplasm. Such changes in Descemet's membrane were observed to varying degrees in four of the seven buttons obtained by penetrating keratoplasty.¶ It is possible, of course, that the two diseases (macular and endothelial dystrophy) were not causally related in our cases, and that the patients simply had two different corneal diseases simultaneously.

As to the specific chemical nature of the material which has accumulated at the sites of collagen disintegration in the corneal stroma, we cannot add much to the suggestion made by Fuchs[9] many years ago that it appears to be mucoid in nature. The newer staining methods which have been devised for the histopathologic demonstration of acid

¶ In one of our eight cases lamellar keratoplasty was performed, and the endothelium could not be studied histologically.

TABLE 2

COMPARISON OF DIAGNOSTIC HISTOPATHOLOGIC FEATURES OF MACULAR AND LATTICE DYSTROPHIES

	Macular	Lattice
Sites of Involvement	Stromal lamellae; corneal corpuscles; endothelial cells	Stromal lamellae
Character of Lesion	Mucoid degeneration	Hyaline degeneration
Birefringence of Affected Lamellae	Markedly decreased or absent	Usually increased
Staining Characteristics of Lamellar Lesions		
Hematoxylin-eosin	Pale gray	Eosinophilic
Masson's trichrome	Faint blue	Red
Wilder's reticulum	Destruction of brown and black fibers	Coarsening of black fibers
Weigert's resorcin fuchsin	Purple	Yellow*
Periodic acid-Schiff	Magenta red	Magenta red
Alcian blue and nuclear fast red	Blue	Red
Rinehart-Abul-Haj	Blue	Yellow*

* Yellow color from picric acid staining in van Gieson counterstain.

mucopolysaccharide do, indeed, reveal these deposits to give intensely positive staining reactions which are not altered by pretreatment of the sections with hyaluronidase or with diastase. The material is, therefore, not hyaluronic acid or glycogen. In all probability it represents one of the sulfated mucopolysaccharides of the cornea which has been freed from its homogeneous relationship to the corneal collagen as a result of the disintegration of the corneal lamellae.

The pathologic anatomy of macular dystrophy as determined from the eight corneal buttons available to us may be summarized as follows:

1. CHANGES CONSISTENTLY PRESENT IN ADVANCED CASES

a. Mucoid degeneration of the stromal lamellae.

b. Accumulation of acid mucopolysaccharide within the corneal corpuscles and between the lamellae.

c. Disappearance of the stromal cells in the more advanced lesions.

d. Accumulation of acid mucopolysaccharide within the endothelial cells.

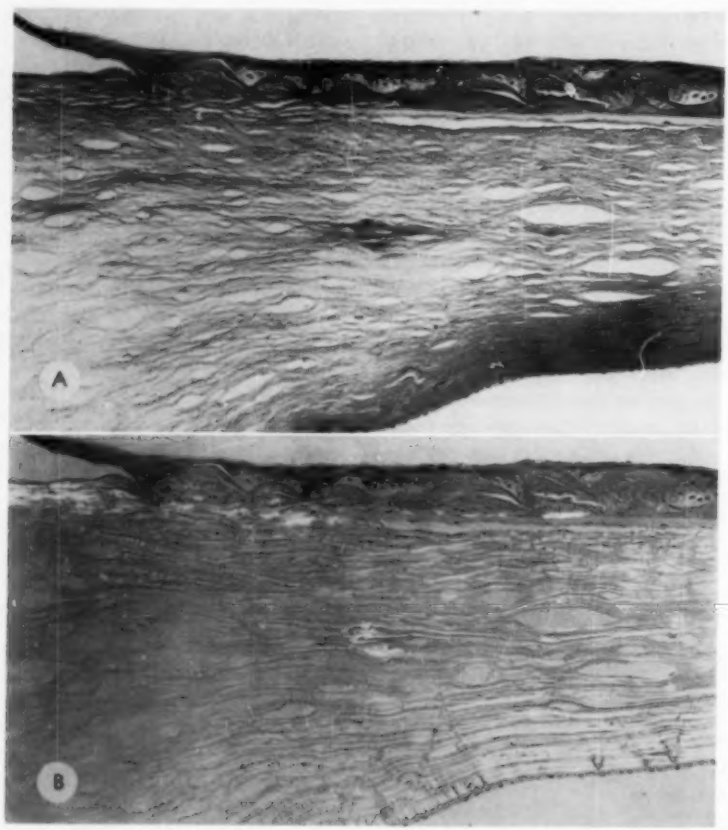

Fig. 9 (Jones and Zimmerman). AFIP Acc. 220210. *Lattice dystrophy.* A focal area of hyalinization of corneal lamellae is present in the center of the field. (A) With ordinary illumination the degenerated stromal fibers reveal a greater degree of eosinophilia than do the uninvolved lamellae. (B) With polarized light the affected fibers appear brilliantly birefringent. (×115.) (Case presented by Dr. F. C. Blodi at 1958 meeting of Ophthalmic Pathology Club.)

Fig. 10 (Jones and Zimmerman). AFIP Acc. 220210. *Lattice dystrophy.* Several affected bundles of corneal fibers exhibit a greatly increased affinity for eosin. (Hematoxylin-eosin, ×305.)

2. CHANGES NOTED FREQUENTLY BUT NOT ALWAYS

a. Elevation and thinning of the epithelium over superficial stromal lesions.

b. Variations in the thickness and staining characteristics of the basement membrane of the epithelium, and of Bowman's membrane.

c. Deposits of hyaline and mucoid material superficial to Bowman's membrane.

d. Changes in Descemet's membrane characteristic of Fuchs' endothelial dystrophy.

HISTOPATHOLOGIC DIFFERENTIATION OF MACULAR AND LATTICE DYSTROPHIES

In addition to the material from patients who had macular dystrophy, we have had available for comparative study five buttons from five patients with lattice dystrophy. Two of these patients were blood relatives. Each of these buttons revealed a distinctive stromal lesion entirely different from those of macular dystrophy. The lesions of macular and lattice dystrophy are compared and contrasted in Table 2 and in the photomicrographs in the color plate. These two hereditary dystrophies are both characterized histologically by focal acellular degenerative lesions affecting the corneal lamellae at all levels. Actually, however, the specific lesions of each have very little else in common, and in several respects their characteristics appear to be opposites. The mucoid degeneration of corneal lamellae in macular dystrophy is associated with a loss of normal birefringence of stromal collagen and a loss of affinity for most stains; by contrast the foci of hyalinization of corneal lamellae in lattice dystrophy are typically more intensely birefringent (figs. 9 and 11) and more intensely stained (figs. 9 to 12) than are the uninvolved corneal fibers. Especially revealing, however, are the widespread accumulations of material exhibiting the staining characteristics of acid mucopolysaccharide in the affected corneal lamellae, in the stromal cells, and in the endothelium of corneas from pa-

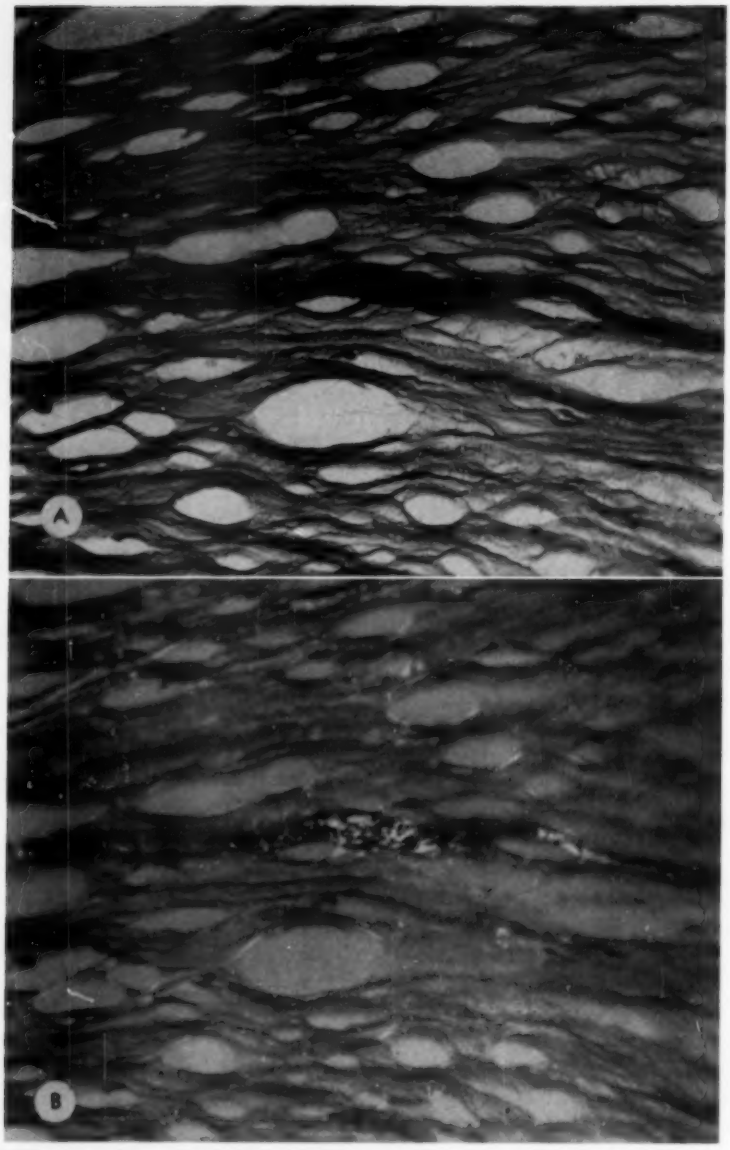

Fig. 11 (Jones and Zimmerman). AFIP Acc. 220210. *Lattice dystrophy.* (A) The deeply stained lamellae in the center of the field appeared bright red with ordinary illumination. (Masson trichrome stain. See color plate K for comparison.) (B) The same field shown in (A) has been photographed with polarized light illustrating that the affected fibers are much more birefringent than the normal corneal collagen. (Blodi's case, ×305.)

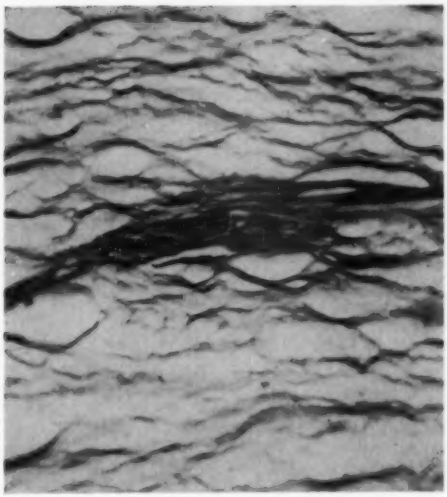

Fig. 12 (Jones and Zimmerman). AFIP Acc. 220210. *Lattice dystrophy.* The Wilder stain reveals a condensation of coarse argyrophilic fibers in the affected lamellae. (×305.)

tients with macular dystrophy and the absence of such accumulations in lattice dystrophy.

Our pathologic studies, therefore, support the contention of Bücklers, which was based on clinical grounds, that the hereditary corneal dystrophies are separate and distinct entities. We have found no case showing a combination of the typical lesions of both lattice and macular dystrophy. In addition, we have found no lesions which we can identify as being "intermediate" or "transitional" between lattice and macular dystrophy.

Franceschetti and Babel[15] also found it possible to distinguish macular dystrophy from the other hereditary corneal dystrophies by histopathologic means.

Future research

Further work is needed to elucidate the fundamental nature of the abnormalities in the hereditary corneal dystrophies, such as electron microscopy, X-ray diffraction, histochemical analysis including that for enzymes, metabolic study of fresh corneal tissue, and examination of the morphologic, biochemical, and metabolic characteristics of the epithelial, stromal, and endothelial cells in tissue culture. By such studies it may be possible to identify for each type of dystrophy a specific metabolic error related to a specific enzyme deficiency or abnormality.

It must be realized, however, that the impetus for such laboratory investigations must come from the clinician. For these studies the surgeon must enlist the aid of experts in electron microscopy, X-ray diffraction, tissue culture technology, enzymology, and histochemistry well in advance of contemplated surgical procedures. A single corneal button can provide satisfactory material for several of these studies, provided the laboratory investigations are well-planned and key personnel alerted to be on hand at the time of surgery.

Conclusions

1. A study of the histopathologic features of eight corneal buttons trephined from patients with macular dystrophy led to the recognition of several consistently present, highly distinctive histologic alterations which included:

a. Mucoid degeneration of the stromal lamellae.

b. Accumulation of acid mucopolysaccharide within the corneal corpuscles.

c. Disappearance of stromal cells in the more advanced lesions.

d. Accumulation of acid mucopolysaccharide within the endothelial cells.

2. A variety of inconstant and nonspecific alterations were also observed:

a. Irregularities in the corneal epithelium.

b. Variations in the thickness and staining characteristics of the basement membrane and of Bowman's membrane.

c. Subepithelial deposits of hyaline and mucoid material.

d. Diffuse and nodular thickening of Descemet's membrane characteristic of Fuchs' endothelial dystrophy.

3. A comparison of the histopathologic characteristics of macular and lattice dys-

trophies has provided ample support for the thesis that they represent separate and distinct disease processes.

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HISTOLOGY AND MECHANISM OF FILTERING OPERATIONS*

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The literature concerning the filtering operation for glaucoma is enormous but most of it is confined to clinical observation. Histologic studies, especially of the functional bleb, are understandably infrequent due to the scarcity of material, but a few have been reported.

Holth,[1] in 1906, presented the first histologic picture after a successful iridencleisis. By 1922[2] he had reported a total of six cases which he had examined histologically. The time between the operation and death or enucleation varied from five months to six years. He presented some fundamental descriptions and principles, describing the blebs as subconjunctival fistula scars, each one looking like a cushion surrounded by an edematous area which can be increased by

* From the laboratory of the Eye-Bank for Sight Restoration, Inc., Manhattan Eye, Ear, and Throat Hospital. This study was aided by grants B-153 and B-1130 from the U. S. Public Health Service.

massage. Histologically the fistula channel is lined by pigmented epithelium from the iris while the inner wall of the bleb is only partially covered with the pigmented epithelium.

In 1931, Holth[3] reported two more histologic studies after successful sclerectomy and one case after an Elliot trephining operation, but the description was not very detailed. Bachstez,[4] in 1914, reported a histologic study of an eye after a successful Lagrange sclerectomy, but again there was no really adequate description.

In 1915, Verhoeff[5] reported one case in great detail, presenting a classic picture of the histology of an eye after successful "sclerostomy."

> The filtering bleb is three mm. in diameter and translucent. The fistula is partially filled with an extremely delicate connective tissue almost free from cells. Within it are numerous irregular, ill-defined, empty spaces, which open directly into the anterior chamber. The edge shows pro feration to form new fibrous tissue and an increase in the number of fixed cells. The bleb consists of highly edematous, delicate connective tissue meshwork containing stellate fixed cells, and closely resembles the stroma of the iris. There are a few blood vessels without inflammatory cells within which occur irregular communicating spaces which at first sight appear empty, but which on close examination are found to be partially filled with a barely visible connective tissue free from fixed cells and continuous with the surrounding stroma. This tissue is even more delicate than that within the fistula. None of the spaces are lined with endothelium. Some of the large spaces extend up immediately beneath the epithelium.

Verhoeff's description of his case is somewhat similar to findings in Figures 11 and 13 in this paper. It is certainly worthwhile to read the whole of Verhoeff's report and study the excellent, detailed picture he presents.

Early attempts at experimental work with animals were unsuccessful, with Ellett[6] reporting, in 1914, that filtering blebs did not form in cats or rabbits. Holth, in 1931, also mentioned that after sclerectomy or iridencleisis in rabbits, the scar tissue would not form a permanent subconjunctival fistula. The only successful animal experiments found in the literature were reported by Spaeth,[7] in 1932, establishing some important fundamental principles of the mechanism of iridencleisis. He noted that in the iris inclusion operation it is the iris epithelium and not the endothelium which lines the wound and maintains the permanent fistula. The iris inclusion tissue gradually undergoes atrophy, establishing a direct communicating fistula between the anterior chamber and the cavity of the bleb.

The theory that the transconjunctival route was the main route of drainage after a successful filtering operation was advanced and confirmed by Herbert,[8] Holth, Elliot,[9, 10] and Lagrange.[11] Seidel[12] demonstrated this route using the fluorescein test. Kronfield[13] recently re-confirmed it and added further proof by chemical analysis.

Other routes were suspected but none have as yet been demonstrated clinically or histologically. In cases where there is no bleb formation, but the tension of the eye is nevertheless controlled after an uncomplicated operation, the existence of one or more other routes must be assumed.

Holth[2] thought that there might be some type of accessory fistulation.

Here it may be appropriate to quote Kronfeld's[14] original description of the clinical appearance of the first two types of functional blebs, for correlation with the histologic picture.

> Type I. A sharply outlined, anemic, polycystic, filtering pad (bleb) consisting of multiple fluid-filled spaces separated from one another by thin, anemic septa. This picture is almost invariably associated with free fistulation or fistulation upon slight pressure, as demonstrated by the Seidel test.
>
> Type II. A flat or slightly elevated zone of succulence, opacity, and anemia in the deep layers of the epibulbar tissues. This zone is located over and around the scleral fistula. By pressure upon the eye the elevation and succulence can be increased.

For several years we at the Eye-Bank have been interested in the early pathology of glaucoma. In our examination of eyes exhibiting such pathology, we have seen a number of eyes in which there was a good

filtering bleb after a filtration operation. Thus the same techniques used in the study of the early histologic changes in glaucoma were used for the study of eyes with filtering blebs, namely serial meridian sections and four stains: hematoxylin and eosin, van Gieson, Verhoeff, and periodic acid-Schiff stains.

We have now studied two specimens after trephining operations, two after iridencleises, one after sclerectomy with iris inclusion, and one after a combined cataract extraction and sclerectomy with iris inclusion.

Filtering bleb after trephining operation with basal iridectomy

Case (EB-106)

The patient, a woman, aged 77 years, had died after a fracture of the femur. She had a known history of glaucoma and a trephining operation but no further details could be obtained.

This eye was studied by means of serial sections and reported as a case of open-angle glaucoma with extensive primary degeneration in the filtration passages of the anterior chamber. There was enough obstruction to cause glaucoma and it was relieved, in our judgment, by the presence of a good filtering bleb after a trephining operation (figs. 1, 2, 3, 4, and 5).

This is a good prominent filtering bleb. The trephine hole was made very close to the edge of Descemet's membrane, slightly more on the trabecular side. The layer of conjunctival epithelium over the bleb is very thin, consisting of one or two layers of cells.

At the center portion of the filtering bleb the epithelium is loose and detached and some epithelial cells have disappeared. This loss of epithelium may be artefact. The basement membrane of the epithelium does not appear over the bleb. Underneath the epithelium there are irregular layers of edematous hyaline and membranous tissue. These are evidently degenerated, hydrated collagen, since they stain lightly and irregularly with van Gieson. The cavity is also filled with degenerated collagen fibers which are distributed more irregularly. Some may be looked upon as partitions.

Beside these there is an amorphous substance mixed with extremely fine fibers which have poor staining affinities and very few cellular elements. This picture is very similar to the collagen degeneration we reported in the chamber angle in open-angle glaucoma.[10]

The posterior wall of the cavity around the trephine hole area is made up of "new connective tissue" derived from the episcleral tissue around the trephine hole. This new tissue covers the anterior third of the thickness of the trephine hole. It consists of two types of tissue—a solid type and a spongy type. Underlying the solid type there is an endothelial lining which is not found under the spongy types (figs. 3 and 4).

In the spongy type of tissue are found coarse fibers which take elastic tissue stain, mixed with finer fibers which have poor affinity for any of the stains used.

Behind the spongy sector there is a layer of granular sludge which contains substances positive for periodic acid-Schiff stain. The posterior portions of both the anterior and posterior lips of the trephine wounds are covered to varying extents by endothelial cells from the iris. This may be due to the collapse of the anterior chamber after the operation and the development of synechias between the iris root and the sclera, which permitted the iris endothelium to grow over and cover the lips of the trephine wound.

Since the anterior wall of the bleb is thin and covered with degenerated epithelium, we would presume that the higher pressure in the anterior chamber caused oozing of the aqueous from the anterior surface of the bleb, constituting a transconjunctival passage.

The degenerative collagen changes also extend along the collagen fibers in the subconjunctival and episcleral regions around the capillaries. The collagen around the capillaries is more or less liquefied, forming perivascular areas of degeneration through which the aqueous may permeate the capillary itself (figs. 1 and 2).

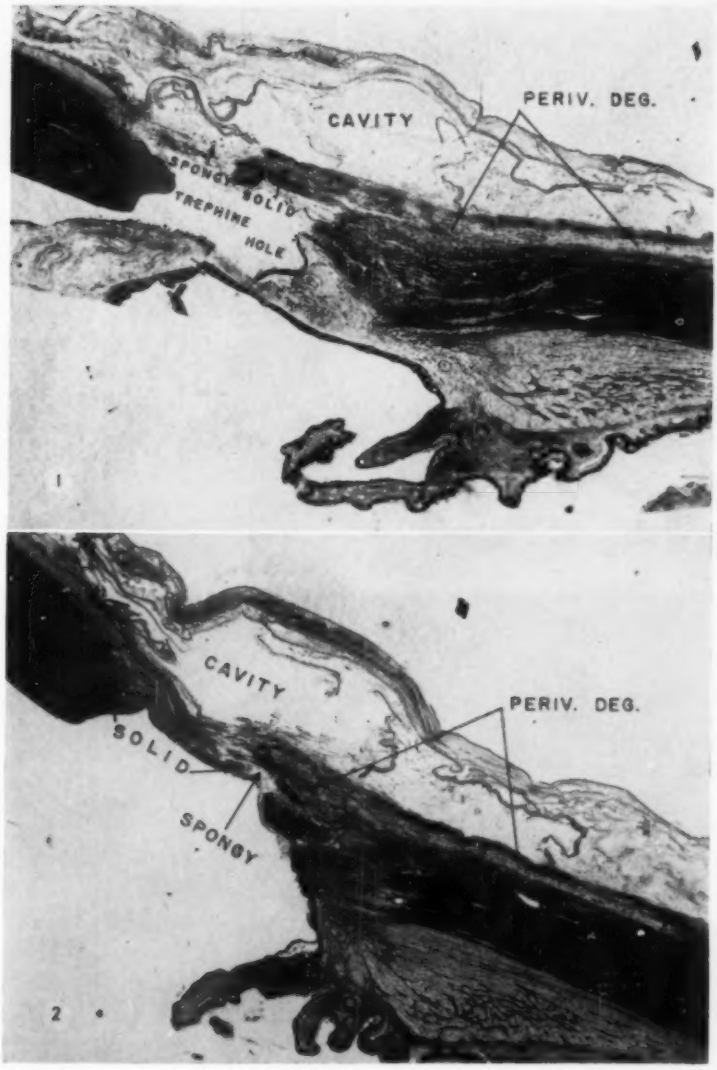
Besides this perivascular route, at the posterior lip of the trephine hole, just behind the new connective tissue, there is a very irregular winding channel lined with endothelium. Reconstruction by serial sections shows that this is actually a new, re-canalized, irregular capillary (figs. 3, 4, and 5 are selected serial sections). This re-canalized capillary established direct communication between the anterior chamber and the deep scleral plexus of veins.

When the collagen at the wound edges is acted upon by aqueous, irregular, wedge-shaped defects are formed and part of the wound is covered by endothelium from the iris. At the same time the endothelium of the capillaries in the area proliferates. Once the two processes meet, a new channel for drainage is formed. Due to the irregularity of the healing processes, the course of this new channel is usually very irregular. This process may explain those cases in which there was lower tension after the operation even though there was no visible bleb formation.

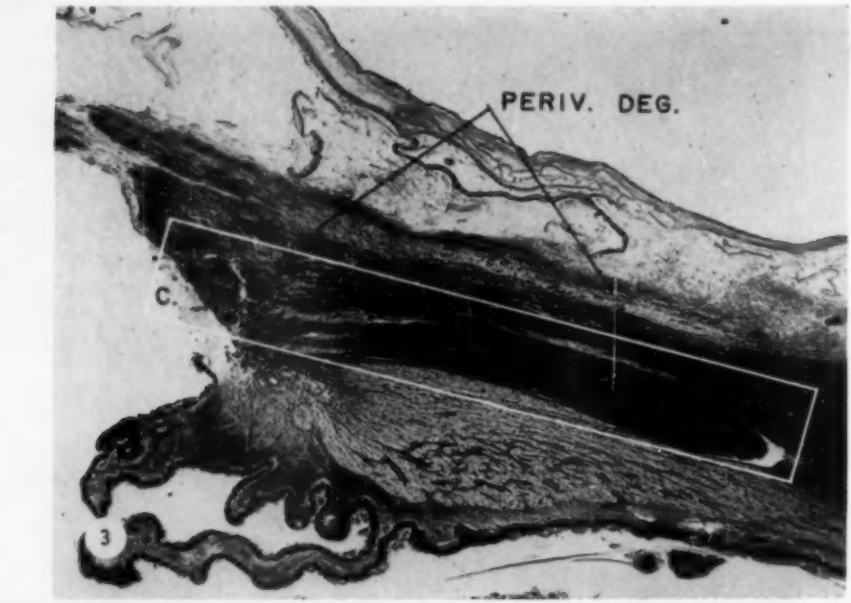
Case 2 (EB-1508, O.S.)

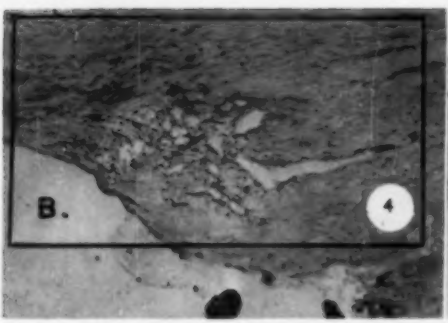
The patient was a woman, aged 76 years, who died of leukemia. She had a known history of glaucoma with high tension. She had had a trephining operation with basal iridectomy, which kept the tension under control until the time of her death, 20 years later.

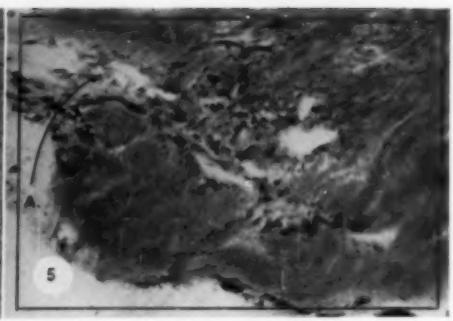
There are some irregular anterior peripheral synechias which are loose and spongy, but it is



Figs. 1 and 2 (Teng, Chi, and Katzin). *Case 1, EB 106, trephining operation.* Van Gieson stain. This specimen exhibits a thin layer of epithelium with broken areas which may be artefact. There are irregular degenerated collagen fibers around the cavity and fine and gross fibers are mixed in an amorphous substance in the inner portion. The solid portion of new connective tissue at the trephine hole has an endothelial lining behind it. The spongy portion consists of coarse elastic fibers. There are also fine fibers which lack staining affinity, and a few cells and pigment granules. Behind the spongy portion there is a sludge collection consisting of granules positive for periodic acid-Schiff stain. Over the episcleral area and posterior lip of the trephine wound there are areas with perivascular degeneration.





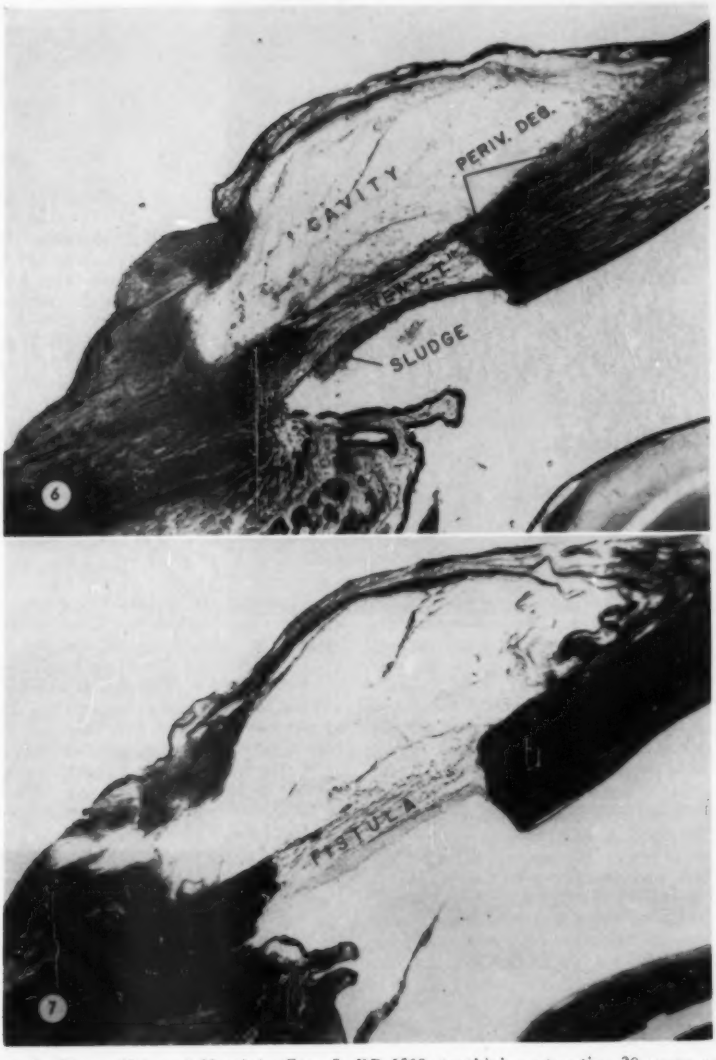


Figs. 3, 4, and 5 (Teng, Chi, and Katzin). *Case 1.* (fig. 3, Van Gieson; fig. 4, periodic acid-Schiff; fig. 5, hematoxylin and eosin.) These three sections were selected from serial sections to demonstrate the winding path of a newly recanalized channel. Area (A) shows the entrance from the anterior chamber; (B) the irregular, winding channel; and (C) the deep scleral plexus. The channel is lined with an irregular layer of endothelium.

doubtful that this type of adhesion could cause much obstruction to the aqueous outflow. The trabecular fibers are essentially normal, except for a portion close to Schlemm's canal. In the collector channels there are definite degenerative changes—both proliferation of endothelium and degeneration of collagen.

In the optic nerve region there is a smooth shallow cupping with a thick cover of glial tissue. The glial tissue and dense collagen tissue replace the degenerated optic nerve fibers in front of the lamina cribrosa. This case had more extensive changes in the optic nerve region than in the chamber angle.

The filtering bleb is a beautiful one (figs. 6, 7, 8, and 9), very similar to the one described in Case 1. The epithelium is very thin and absent at the central portion. The basement membrane is very irregular, appearing double in some places and totally absent in others. Under the epithelium there are irregular layers of connective tissue, consisting mostly of collagen which exhibits varying degrees of degeneration, with the less degenerated portion at the outer layer. Except for a few thick collagen fibers which are irregularly distributed and clinically may look like partitions, the inside of the cavity is filled with degenerated collagen appearing in the form of fine lax fibers and an



Figs. 6 and 7 (Teng, Chi, and Katzin). *Case 2, EB-1508, trephining operation 20 years ago.* (Fig. 6, Verhoeff stain; fig. 7, Van Gieson.) Thin irregular epithelium. The lining of the cavity of the bleb is composed of degenerated collagen in the form of coarse and fine fibers and an amorphous substance is found at the center. New connective tissue has formed around the edges of the cavity, particularly at the area of the trephine hole. This new connective tissue is spongy and consists of coarse and fine elastic fibers. There is no solid collagen fiber formation, but there are fine, irregular fibers without staining affinity. Behind this is a sludge collection. There is no cellular lining either anteriorly or posteriorly to the new connective tissue. Some perivascular degeneration can be seen in Figure 6.

amorphous substance mixed with aqueous.

Part of the anterior lip of the trephine wound is covered by epithelium from the prolapsed ciliary body. There is also new connective tissue growing from the episclera toward the wound. The new connective tissue consists of fine irregular fibers and coarse "elastic fibers." We refer to these as "elastic fibers" because they are positive for Verhoeff's elastica stain, even though we are aware that regeneration of elastic fibers in wound healing is

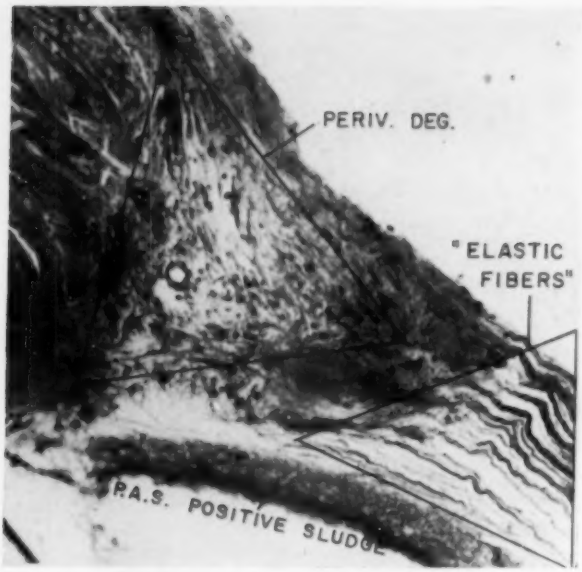


Fig. 8 (Teng, Chi, and Katzin). *Case 2.* (Periodic acid-Schiff stain.) Perivascular degeneration at the edge of the trephine wound. Coarse elastic fibers are mixed with fine fibers in the new connective tissue which fills the trephine hole. The sludge collection is positive to periodic acid-Schiff stain.

still an unsolved problem.

There are very few cells. On the posterior surface of this new loose connective tissue septum there is a noncellular layer made up of granular sludge which is periodic acid-Schiff positive. Gonioscopically, this may have looked like a dome-shaped white roof; it may have appeared solid and smooth or like lacy white tissue, which may form a netlike structure within the dome as described by Kronfeld.

Perivascular degeneration is found in the subconjunctival and episcleral regions. Some perivascular degeneration is found in the unhealed wounds of sclera (fig. 8) and the ciliary body (fig. 9). These are due to the fact that the wounds are not covered by a protective cellular membrane, and are thus exposed to the aqueous. The exposure to aqueous causes degeneration of collagen and creates perivascular degenerative areas.

The iridectomy wound looks like a fresh one, without evidence of fibrosis (fig. 10). The cut end of the blood vessels are not constricted and there

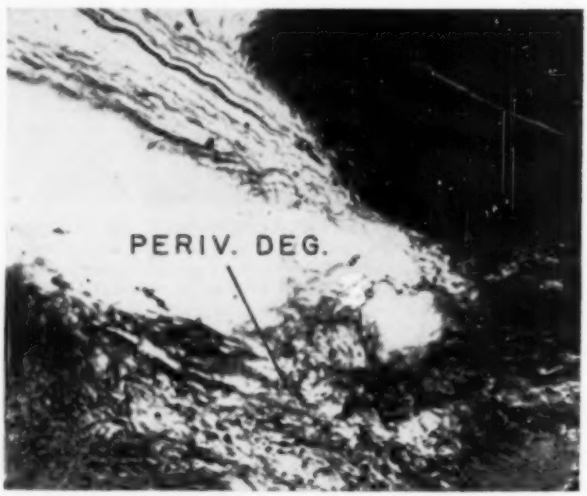


Fig. 9 (Teng, Chi, and Katzin). *Case 2.* (van Gieson stain.) Degeneration of collagen has formed a perivascular area of degeneration in the ciliary body at the root of the iris where the trauma of the iridectomy has disturbed the endothelial covering.

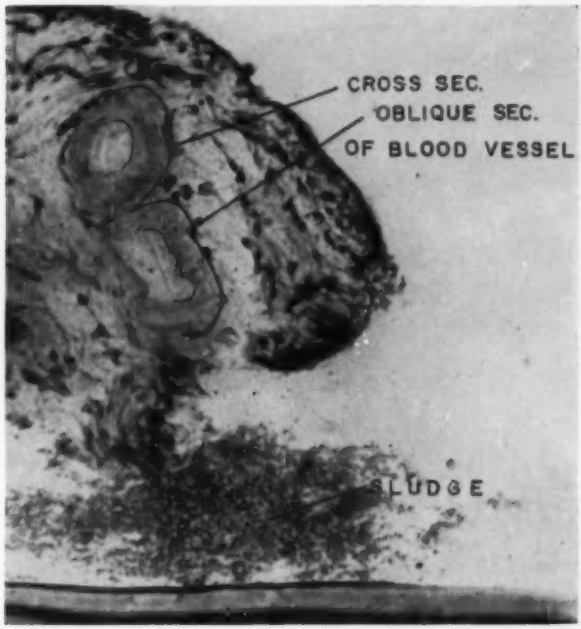


Fig. 10 (Teng, Chi, and Katzin). *Case 2.* (Iridectomy wound. Verhoeff stain.) In this oblique section of the blood vessel there is no constriction of the lumen near the wound due to the thick, firm collagen wall. The wound appears like a fresh one, without fibrosis.

is mild perivascular degeneration of collagen. There is also a collection of sludge at the cut end. This may indicate that there is a drainage process going on at the iridectomy wound.

The filtering blebs in these first two cases are similar to Kronfeld's Type I filtering bleb and to the case reported by Elliot.

Filtering bleb after iridencleisis

Case 3 (EB-4273, O.D.; 4274, O.S.)

This history was obtained through the kindness of Dr. Edward J. Bassen, New York. The patient was a woman who had died at the age of 70 years, having had a history of glaucoma. An iridencleisis had been done in 1923 and she died of arteriosclerosis and heart disease in 1956. Since the third postoperative year, the patient had complained occasionally of tired, watering eyes with occasional headache. The tension, however, was never above 25 mm. Hg and usually below 20 mm Hg. Vision continued good until the last seven years when it grew poorer, due to lens changes. The filtering bleb was small, but of good shape. There were no changes in the fundi or visual fields after the operation. She had two functional filtering blebs for 33 years.

The anterior chamber region, the external portion of the trabecula, and especially the area of Schlemm's canal and the collector channels exhibit moderate primary degeneration and adhesion with collection of pigment granules. There are extensive postoperative anterior peripheral synechias.

The optic nerve was essentially normal in both eyes.

The filtering blebs are good in both eyes (figs. 11, 12, 13, 14, 15, and 16). The incisions in each case were made at the limbus, but more on the corneal side. Each bleb consists of a thin, but intact epithelial lining and subconjunctival tissue fused with episcleral tissue. The conjunctival epithelium is thinner than normal. Below the epithelium there is loose edematous connective tissue which includes degenerated collagen fibers and abundant coarse elastic fibers. There is extensive perivascular degeneration around the vessels of the subconjunctival and episcleral regions and these form the principal route of drainage.

The iris inclusion leads from the anterior chamber through a fistula into the cavity. The iris rests between the subconjunctival and episcleral regions. The cavity is larger in the left eye (figs. 11 and 12) than in the right (figs. 14, 15, and 16). There is no continuous cellular lining of the cavities. The stroma of the iris inclusion tissues is mostly degenerated and forms a communicating path from the anterior chamber into the cavity. In both eyes portions of the fistula are lined with endothelium from the iris, and part of the posterior wall in the right eye is lined with iris epithelium. In the right eye the fistula is straighter due to the greater degree of degeneration of the iris stroma in that eye.

Another difference between the two eyes is that the fistula in the right is filled with albuminlike

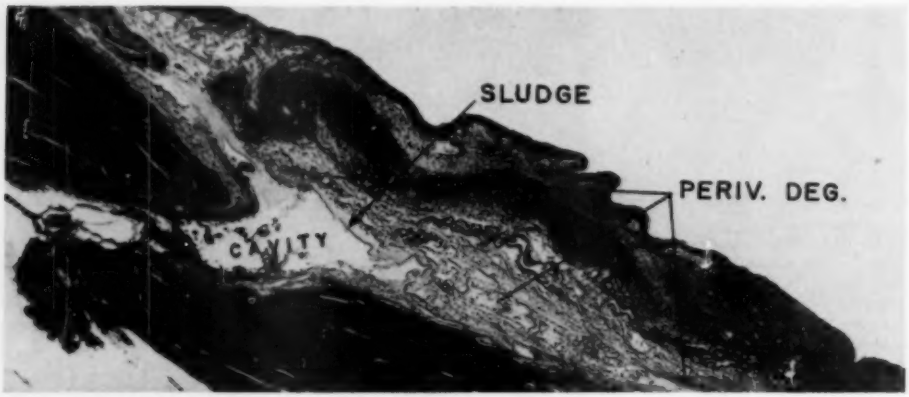


Fig. 11 (Teng, Chi, and Katzin). *Case 3, EB-4274.* Filtering bleb after iridencleisis 33 years ago. (van Gieson stain.) This bleb has a smaller cavity and a much thicker outer wall.

substance and at its inner orifice, on the level of Descemet's membrane, there is a collection of sludge which is positive to periodic acid-Schiff stain (fig. 16). In the left eye the channel leads to a larger cavity in the episcleral region and the sludge has collected at the inner surface of the cavity (fig. 12). This resembles the collection of sludge in the eyes which had trephining operations. The bleb is very similar to the Type II filtering bleb described by Kronfeld.

Filtering bleb after Lagrange operation with iris inclusion

Case 4 (EB-3998)

This case and the detailed history were obtained through the kindness of Dr. L. Benjamin Sheppard of Richmond, Virginia.

The diagnosis of open-angle glaucoma was made in 1940. For 13 years the tension was never very high (20 to 28 mm. Hg Schiøtz), but in March, 1954, the tension in the right eye went up to 39 mm. Hg. Dr. Sheppard did a Lagrange operation with iris inclusion on the right eye. The operation successfully lowered the tension to 17 to 20 mm. Hg. After the operation, there was rapid development of a cataract and a cataract extraction was done through a corneal incision to avoid the functioning bleb. This improved the vision from 20/200 to 20/70 and the tension remained around 20 mm. Hg until the time of the patient's death in July, 1955, from carcinoma of the stomach at the age of 85 years. The eyes were donated to the eye-bank. At this time the filtration bleb was 16 months old.

Histologic study shows that the anterior lip of the sclerectomy wound is partially covered by Descemet's membrane (fig. 17), while the posterior lip is partially covered by endothelium from the iris inclusion (fig. 18). The iris inclusion material is slightly degenerated and overlies a degenerated area of sclera.

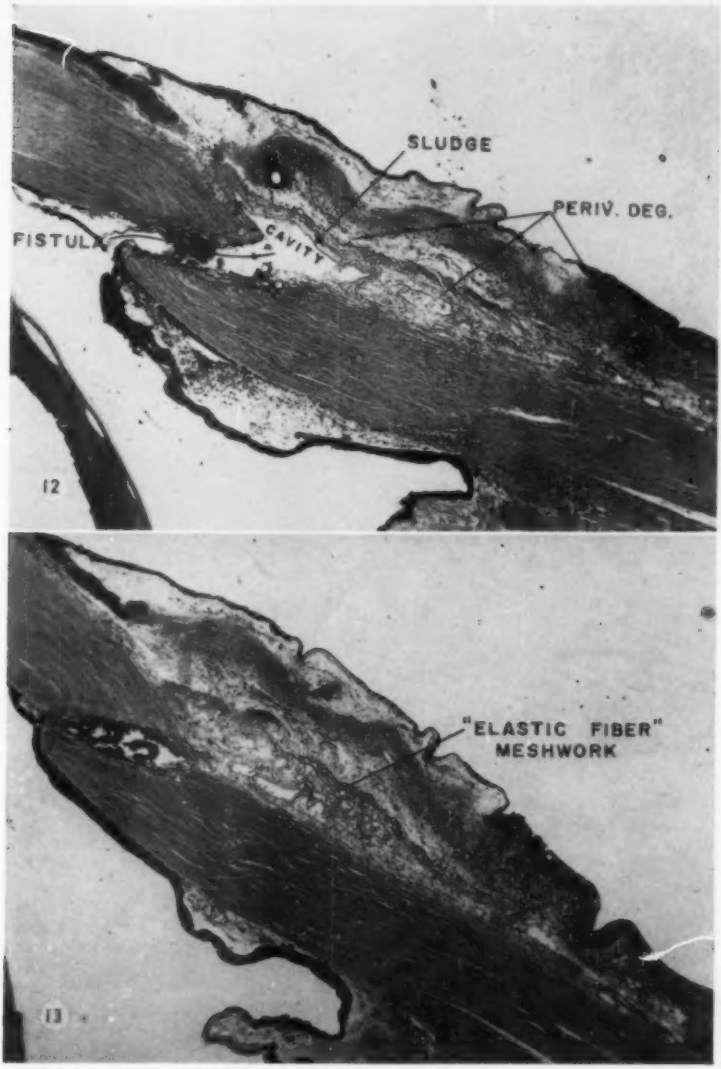
The surface of the posterior lip of the scleral wound is fluffy due to the action of aqueous. In one place the degenerative change extends along a deep scleral vessel, forming an area of perivascular degeneration for accessory drainage of aqueous (fig. 18).

The cavity of the bleb is long and narrow. Its anterior wall is composed of epithelial and subconjunctival tissues. It is a thicker wall than usual, with much less hydration and degeneration and there are more collagen components. The inner surface of the anterior cavity wall has no cellular lining except for some pigment granules, and this portion therefore exhibits edema and the typical changes seen when connective tissue is in direct contact with the aqueous. The posterior wall of the cavity is lined with episclera. Posterior to this narrow cavity, but still along the episcleral area, there is a long, narrow zone where degeneration of connective tissue has resulted in an area of degenerative perivascular spaces, which form a new drainage system for aqueous.

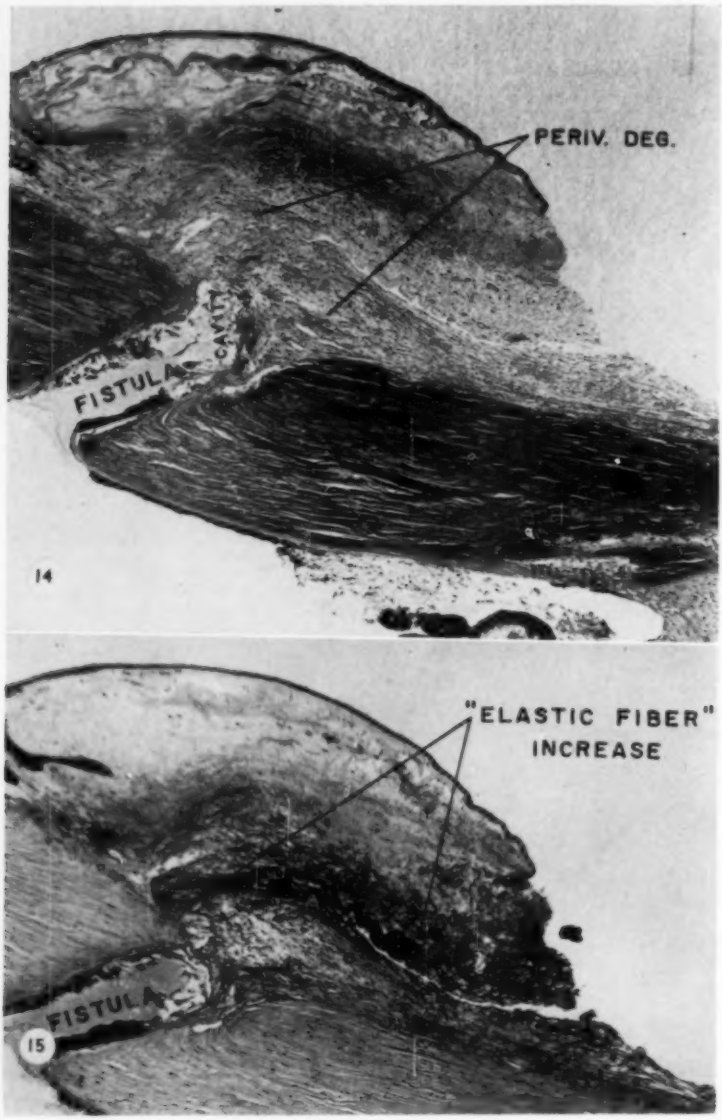
Since the anterior wall of the cavity exhibits less change, we can presume that there would be very little transconjunctival drainage. The main route of aqueous drainage is through the extensive episcleral and subconjunctival areas of perivascular degeneration, or perhaps in part, through the new accessory route in the sclera (fig. 18).

This bleb is only 16 months old. There are very few elastic fibers as compared with older blebs, probably indicating that elastic fibers are slower to form. This filtration bleb looks healthier than the bleb after a trephining operation, offers less chance of infection, and less chance for traumatic rupture of the bleb.

Clinically this may be a Type II filtering bleb according to Kronfeld's[14] and Pillat's[16] classification, but it has a wider fistula and a larger cavity, and presumably a greater



Figs. 12 and 13 (Teng, Chi, and Katzin). *Case 3.* (Fig. 12, hematoxylin and eosin; fig. 13, Verhoeff stain.) The epithelium is thinner than normal, consisting of only two to four layers of cells but there is no break in the surface. The newly formed connective tissue in the subconjunctival region contains mature collagen fibers, while tissues around the cavity show degeneration of collagen and increase of coarse elastic fibers mixed with fine fibers of collagenous origin. There are many perivascular areas of degeneration over this region and a thin layer of sludge behind the spongy anterior wall of the cavity. The winding course of the fistula is due to irregular degeneration of the iris inclusion material. Some sectors still show a considerable remnant of degenerated iris tissue. The fistula is lined for the most part with iris endothelium. The caliber of the passage is smaller than that formed by trephination.



FIGS. 14 and 15. (Explanation given under Fig. 16)

capacity for drainage than the bleb resulting from an iridencleisis.

CASE 5 (EB-3860)

This case was obtained through the kindness of Dr. Wendell Hughes of Hempstead, New York. The patient was first seen by him on May 3, 1954, with a history of poor vision in the right eye for the previous five years, and in the left eye for two years. The vision in the right eye had always been poorer than in the left.

Best vision at the first visit was finger counting at six feet, O.D., and 20/100, O.S. Pupils were normal and there were lens opacities in each eye.

The fundi were indistinctly seen under cyclogyl mydriasis and no pathologic process was noted.

Tension was 37 mm. Hg (Schiøtz) in the right eye, and 30 mm. Hg in the left eye. Fields showed 10 to 15 degrees of constriction in each eye in the 3/330 isopter.

On May 5, 1954, a cataract extraction and filtering operation were performed at the same time.[11] In one month the vision was 20/30 and it later improved to 20/20. There was a good filtering bleb above. Tension remained low for 11 months, until the patient's decease April 3, 1955, of acute inflammation of the large bowel with ulceration of the mucosa. The filtering bleb was then 11 months old.

Histologically the angle of the anterior chamber shows some rather extentive anterior peripheral synechias on the side of the iris inclusion.

On the other side of the chamber angle there are many irregular synechias, some covering the whole trabecular surface.

The external portion of the trabecula, especially, the inner wall of Schlemm's canal and the collector channels in that area, exhibits some proliferation of endothelium and adhesion, but it does not seem extensive enough to cause a rise in tension. The rise in tension would therefore seem to be due to the double factors of primary degeneration and anterior peripheral synechias.

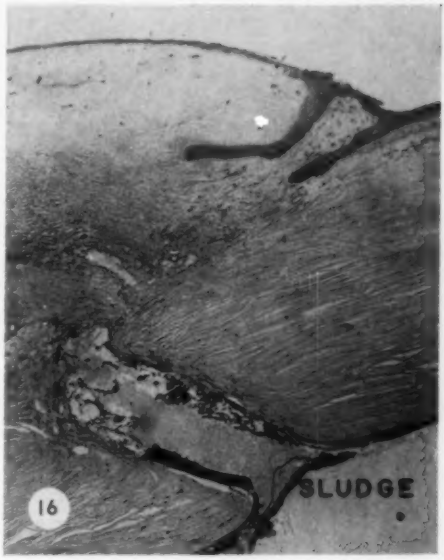
The optic nerve is essentially normal except for some perivascular change.

The structure of the filtering bleb (figs. 19, 20, 21, and 22) is much like that of a trephining operation, except that the gap of the wound and the size of the bleb are larger. The wall of the cavity of the bleb is very thin, probably due to the large, gaping nature of the wound and the consequent free access of aqueous to the collagen. It is a good, functional bleb, the aqueous filtration being mainly by the transconjunctival route.

The anterior lip of the wound is covered by new connective tissue, formed from the episcleral and subconjunctival tissues where there is extensive perivascular degeneration.

The posterior lip, at the central portion is covered by Descemet's membrane and the rest is covered by iris epithelium. The iris inclusion overlying the sclera exhibits very little degeneration. Its structures are mostly well preserved. The epithelium of the anterior wall of the bleb is very thin, consisting, in some sectors, of only one or two layers of cells. The basement membrane of epithelium is preserved in only a few areas. The subconjunctival and episcleral tissues are very edematous, with little staining affinity. The posterior surface of the anterior wall appears to be in direct contact with the aqueous over a wide area. The anterior wall is very edematous and consists of degenerated collagen and some coarse elastic fibers. There is no lining of cells on the posterior surface.

At the junction of tissues along the subconjunctival and episcleral regions there are abundant capillaries with perivascular degeneration which may indicate that there are secondary passages for the absorption of aqueous (fig. 21).



Figs. 14, 15, and 16 (Teng, Chi, and Katzin). *Case 3. EB-4273, OD.* (Fig. 14, hematoxylin and eosin stain; fig. 15, Verhoeff stain; fig. 16, periodic acid-Schiff stain.) This filtering bleb is similar to that in the left eye, except that the cavity is smaller and the fistula is filled with an albuminous substance. There is a deposit of periodic acid-Schiff positive substance (sludge) behind it.

Dr. Hughes[17] has performed this type of operation in 29 eyes and in only one case was he unsuccessful in lowering the tension and improving the vision.

This is a typical Type I filtering bleb, with an even larger passage for aqueous than the trephining operation provides.

Discussion

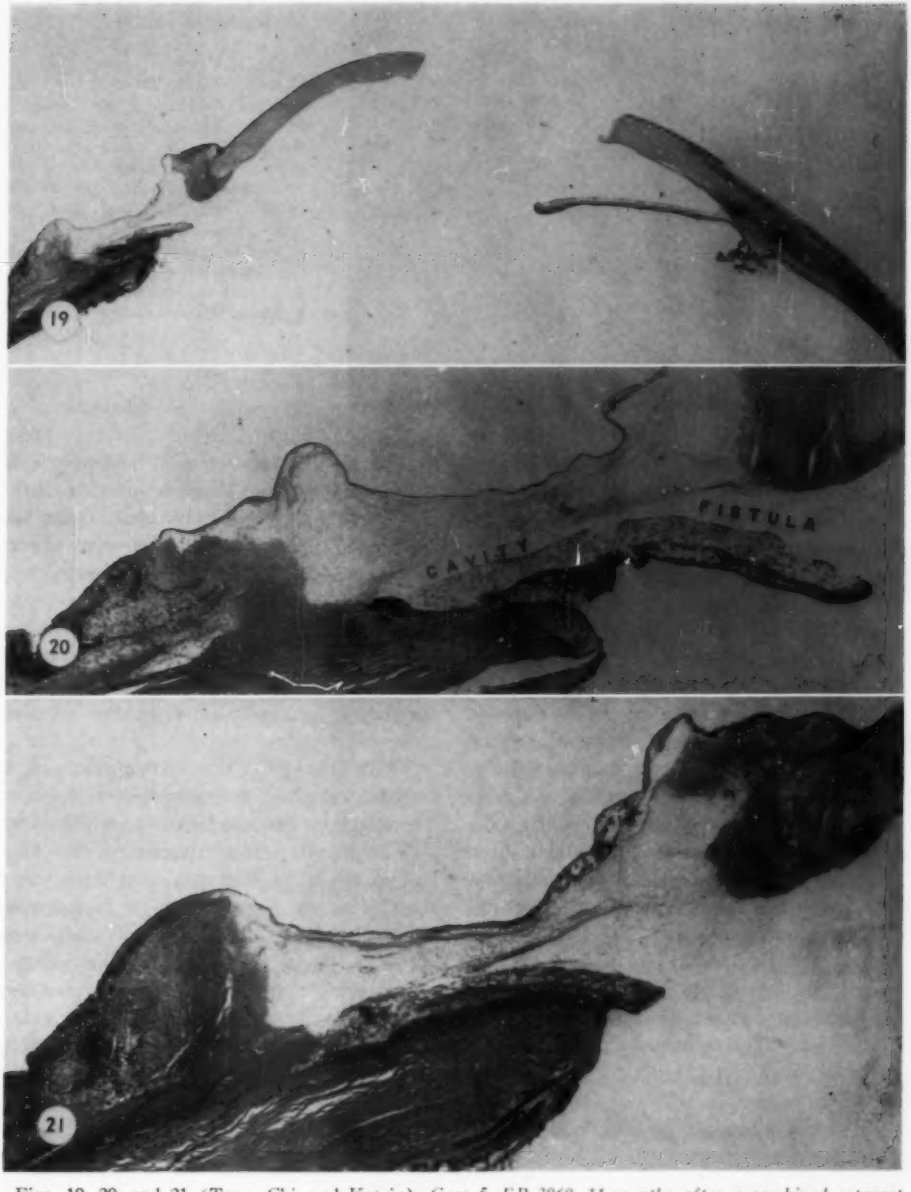
The demonstration of the action of aqueous on connective tissue and especially on collagen may be of the utmost importance, not only because of its importance in the cause of open-angle glaucoma and in the formation of a functional filtration bleb after filtering operations, but because this action may help to explain many physiologic and pathologic phenomena in the eye.

We would like especially to stress the importance of the protective effect of a lining of endothelial or other cells. After break-up

Figs. 17 and 18 (Teng, Chi, and Katzin). *Case 4. EB 3998, 16-month-old bleb after sclerectomy and iris inclusion operation* (fig. 17, above, Periodic acid-Schiff stain; fig. 18, at left, hematoxylin and eosin). The separation of the epithelium on the surface of the conjunctiva is an artefact. The cavity has a much thicker outer wall due to the increase of new connective tissue in the subconjunctival and episcleral regions. There is no cellular lining in the cavity and there is mild degenerative change in the tissue around the inner surface of the cavity. Over the episcleral region behind the cavity there is a long, narrow area with perivascular degeneration. At the posterior lip of the sclerectomy wound there is no cellular covering and there is consequent destruction of the collagen. In one place this area of deteriorated collagen leads into a perivascular area of degeneration (fig. 18) around a deep intrascleral plexus. The fistula of the sclerectomy is small and winding, and only the inner margin is covered by endothelium and Descemet's membrane. The big mass of undegenerated iris inclusion may act as a door-jamb, holding open the passage for aqueous.

and loss of a protective lining, the degenerative process starts, because the aqueous has direct access to the underlying tissues. Perhaps partially due to hydration, the collagen fibers swell, disperse, and lose their ground substance and supporting framework. The usual staining affinity is lost and finally there are only fine irregular fibers and an amorphous substance left. The end-result is a liquefaction of the collagen. There is no definite direct effect on the cells—the apparent loss of cells is probably due to the loss of the supporting substances.

The shape of the fibroblasts is changed, perhaps due to this lack of supporting collagen, and they become free cells in the aqueous. Since there is still proliferation of cells at the edge of the wound, it would seem that aqueous has only a mildly deleterious effect on fibroblasts.



Figs. 19, 20, and 21 (Teng, Chi, and Katzin). *Case 5. EB-3860, 11 months after a combined cataract extraction and sclerectomy with iris inclusion.* (Hematoxylin and eosin stain.) The very wide fistula allows greater contact with aqueous. There are more pronounced collagen changes resulting in a greater transconjunctival passage of aqueous. The proliferation of connective tissue at the edge of the wound is very evident and some perivascular areas of degeneration have formed at the subconjunctival and episcleral regions.

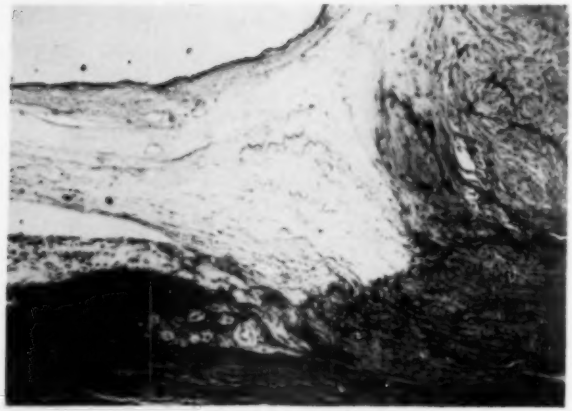


Fig. 22 (Teng, Chi, and Katzin). *Case 5.* (Verhoeff stain.) At the edge of the cavity there is proliferation of connective tissue from the episcleral and subconjunctival regions. There is practically no mature collagen in the wall of the cavity, but there are plenty of coarse and fine elastic fibers. This is because contact with the aqueous has destroyed the old collagen and prevented the deposit of new collagen but the old elastic fibers persist and new elastic fibers have formed.

Fibroblasts may not be able to live long in an aqueous medium, although this is known to be a favorable environment for the growth of many kinds of cells. In 1955 we reported[15] our suspicion that aqueous had a deleterious effect on collagen when there was a defect of the trabecular endothelium; and that this was a possible cause of primary degeneration and subsequent blockage of the filtration passages in some cases of open-angle glaucoma.

Snell,[18] (1956), in a study of wound healing of the iris, reported that granulation tissue implanted into the anterior chamber would not grow and would not cover an iris wound resulting from diathermy. He questioned whether this was due to inhibited proliferation or inhibited cohesion due to the effect of aqueous.

Also in 1956 Kornblueth and Tenenbaum,[10] in their tissue-culture work, noticed the inhibitory effect of aqueous on fibroblasts. The outgrowth was very limited and the migrating cells exhibited a rounding-off and early degeneration.

All of these reports are actually describing the deleterious effect of aqueous in different ways. The exact physicochemical effect of the aqueous on collagen is not understood, but it may be due to hydrolysis, solubilization, or enzyme action. Further investigation of this question is fundamental.

The importance of the protective sheath of endothelial cells has been recently demonstrated in this laboratory when pieces of sclera were implanted into the anterior chamber of rabbits' eyes.[20] In most cases the pieces of sclera became encapsulated with endothelial-like cells, and the connective tissue lived unchanged for up to 13 months; but in the cases where the implant was not so encapsulated the sclera showed a collagen degeneration similar to that observed in open-angle glaucoma and in the filtering bleb.

There is evidence that vitreous, as well as aqueous, may have the same deleterious effect on collagen. Our observations on this point will be reported in a separate paper.

The newly proliferated connective tissue that forms at the edge of the trephine wound again demonstrates the effect of aqueous on collagen, but in a reverse way. The presence of aqueous at this site is shown by the fact that there is no deposit of collagen unless there is an epithelial or endothelial lining over the connective tissue. After the formation of this lining there is deposition of collagen. As the connective tissue becomes more solid, it becomes much less permeable to aqueous.

There is also evidence that whether the bleb develops as a Type I or Type II bleb, as described by Kronfeld, depends upon the amount of aqueous action on the exposed collagen of the subconjunctival and episcleral connective tissue.

The resistance of elastic fibers to the deleterious effect of aqueous was noted in our studies on primary degeneration of the chamber angle. In filtering blebs it is demonstrated even more definitely. The abundance of elastic fibers in old blebs and the comparative scarcity in the newer blebs indicate that the formation of new elastic fibers is a slow process. This needs further study to clear many doubtful points.

Mechanism of formation and function of a filtering bleb after trephining operation

Immediately after the operation there is a great rush of aqueous into the subconjunctival cavity. This immediately starts the processes which result in degeneration of the collagen of the subjunctiva and episclera. At the same time there is a healing process starting around the edges of the cavity as new connective tissue is formed. The result is a large cavity with a thin wall.

Due to the thin, loose texture of the tissue of the anterior wall of the cavity and to the defect in the epithelium and basement membrane, there is a natural oozing of aqueous through the wall.

This transconjunctival route of filtration may not have a sound physiologic basis and, with the frequency of defects in the epithelium, this area is very liable to infection and to accidental rupture. Nevertheless, we have one case of 33 years' duration, which was still in good condition at the time of the patient's death, and there are many cases observed clinically for longer periods, where the filtering bleb is still functional and without complication. Before one can pronounce on the advantages and disadvantages of this type of operation, a thorough analysis of a great number of uncomplicated, controlled cases would have to be made.

The filtering bleb resulting from a trephining operation evidently gives a greater capacity for aqueous drainage than that resulting from iridencleisis. This is consistent with Dunnington's[21] clinical observation that the trephining operation is more effective in advanced stages of open-angle glaucoma, where the tension is very high.

As to the new connective tissue which forms from the episcleral region at the trephine hole, there seems to be no way to stop the growth altogether, but our observations seem to indicate that if there is no cellular lining on the posterior surface, there will be no deposit of collagen, and a good, filtering structure will be maintained.

This picture of new connective tissue has been observed and discussed by Haas, McGarry, and Kronfeld,[14, 22] who examined functional blebs by gonioscopy. They described it as dome-shaped, lacy, white tissue at the trephine wound. This type of new connective tissue seems very accessible to aqueous. Perhaps this report of the histology of such wounds can answer some of the questions they raised.

The histologic picture, as we interpret it, shows that aqueous not only has a deteriorating effect on mature collagen, but it also works to prevent fibroblasts from forming collagen. The resistance of elastic fibers to the deleterious effect of aqueous may explain the presence of elastic fibers in filtering blebs and its abundance around the episcleral and conjunctival regions.

The lining of the trephine wound, indeed the whole surface of a trephine wound or other scleral wound, ideally, should not be covered by any endothelium or epithelium. We have observed that where there is no such covering, aqueous acts upon the collagen tissue, which deteriorates and thus permits the formation of accessory filtration routes. These may be in the form of recanalization or in the form of perivascular filtration routes.

Aqueous action may even explain the occurrence of spontaneous fistulation in the neighborhood of a perforating vessel, or a spontaneous accessory channel through fistulation formation at the site of a trephine hole, as described by Elliot.[9, 10]

Except for the danger of hemorrhage, it might be advisable to place the posterior edge of the trephine wound at the trabecular re-

gion, which is very rich in blood vessels, thus providing a better chance for direct recanalization or filtration through a perivascular route. Sometimes the trephine wound may even open directly into a healthy and patent portion of the diseased filtration passage and re-establish the drainage system.

Mechanism of formation and function of filtration blebs after iridencleisis

Our histologic observations support Kronfeld's report that the filtering bleb in iridencleisis is usually Type II.

The mechanism we observed is very much like that reported by Spaeth. The purpose of the iris inclusion is to provide a surface lining for the scleral wound, or the fistula, from the endothelium or epithelium of the iris and to prevent the extension of Descemet's membrane or the endothelium of the cornea over the wound, because these structures form a less permeable barrier.

The second object of the iris inclusion is, after its degeneration, to provide a pathway, or fistula, for the aqueous between the anterior chamber and the subconjunctival cavity.

As Holth pointed out, the iris inclusion prevents immediate, sudden fall of tension, and allows the anterior chamber to reform sooner after the operation, since some degree of tension is preserved. Then the stroma and other tissues of the iris begin to show degeneration, probably due to pressure, the consequent compression of iris circulation, and the effect of the aqueous. After degenerating, these tissues allow seepage of aqueous through the irregular passage. Thus, during the first stage, immediately after the operation, the iris inclusion limits the passage of aqueous into the cavity and diminishes the deleterious effect of the aqueous on collagen. As a result, a healthy cavity with a comparatively thick wall is formed.

Around the blood vessels of the subconjunctival and episcleral regions are pronounced degenerative changes due to aqueous filtration into the interstitial tissues. Thus, in the subconjunctival and episcleral regions a distinct perivascular aqueous filtration route is formed. In cases with high tension, more aqueous enters the cavity and this may cause the degenerative changes to extend up immediately beneath the epithelium. If these changes are extensive, a small local transconjunctival filtration route may be established.

Histologic evidence indicates that the drainage capacity of this type of bleb is much smaller than that of the functional trephine bleb. This corresponds with Dunnington's clinical observation which indicated that iridencleisis was the operation of choice in mild cases of open-angle glaucoma.

Another advantage of the iridencleisis is the thicker wall of the cavity is less liable to infection and to rupture due to trauma.

The histologic picture resulting from the sclerectomy with iris inclusion is very similar to that of the iridencleisis, except that the first provides a larger cavity with a greater capacity for aqueous drainage.

Conclusions

There are three main routes of aqueous drainage which can be created by filtering operations: (1) the transconjunctival route; (2) the route through areas of perivascular degeneration; and (3) direct new recanalization.

The development of the first of these, the transconjunctival route, is evidently due to the greater amount of aqueous in contact with the connective tissue. As the collagen degenerates and becomes more permeable, the epithelium and basement membrane of the conjunctiva are also affected, so that there is oozing out of aqueous through the bleb.

Perivascular degeneration occurs when the aqueous affects the collagen around the blood vessels, especially the capillaries, thus creating loose perivascular degeneration which facilitates filtration. The episcleral and subconjunctival regions are usually rich in blood vessels and it is there that the perivascular routes of drainage develop.

Direct new recanalization is especially

likely to develop in cases where a scleral wound is close to the trabecular region. There are plenty of capillaries in this area. Due to the action of the aqueous, irregular wedge-shaped defects are covered by proliferation of endothelium, and, at the same time, there is proliferation of endothelium from the cut ends of the capillaries. When these two proliferated tissues meet, a new drainage channel lined with endothelium is formed. The new channels may be very irregular, but they are open passages and therefore more effective than the other routes. This type of route formation may explain many cases in which there is no visible bleb after a filtering operation, but the tension is nevertheless lowered. The technique of producing this type of filtration should be further investigated.

Another possible route of aqueous filtration, not demonstrated in this report, could be the re-opening of the drainage channels by cutting into a patent portion of Schlemm's canal or the collector channels near an area of obstruction.

Postoperative function examination

If a bleb is well formed after a filtration operation, one always finds that it is functional and the tension is lower. In these cases the filtration may be through either the transconjunctival route or through the perivascular degenerative areas. But in those cases in which the operation succeeds in lowering the tension without a visible bleb formation, the filtration may be due either to the establishment of a direct route or to perivascular degeneration.

The postoperative functional examination is first an external one. Kronfeld's description of the external appearance of different types of postoperative appearance is very valuable and should be carefully reviewed.

The best test to see whether there is filtration through the transconjunctival route is the Seidel test with a fluorescein drop on the bleb, application of pressure, and observation of the flow of aqueous.

Gonioscopic examination of the wound of a filtration operation gives a very limited amount of information, as Kronfeld, Troncoso and Reese,[23] and Devoe[24] agree. As we learn more about the pathology and mechanism of the operation, we may come to understand the gonioscopic picture better.

At present the most valuable tests for patency, where there is no visible bleb, may be tonography and Goldmann's fluorescein test.

Our findings tend to support the mechanical theory of the filtration operation rather than the neurovascular theory suggested by Weekers,[25,26] Devoe, and Meyer.[27]

Summary

Histologic studies of six successful filtering operations are reported, two specimens after trephining operations, two after iridencleisis, one after sclerectomy with iris inclusion, and one after a combined cataract extraction and sclerectomy with iris inclusion.

The direct effect of aqueous on connective tissue, especially collagen, is re-emphasized and discussed in connection with the mechanism of filtration.

Three ways in which functional filtering blebs are formed and operate are described: the transconjunctival route, the perivascular routes, and direct new recanalization.

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SURGICAL MANAGEMENT OF PRIMARY GLAUCOMA

A DESCRIPTION OF A NEW TECHNIQUE

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It can be said that nowadays in primary glaucoma, surgical treatment is considered most efficacious for the mechanical narrow, closed-angle type. Medical treatment (miotics or inhibitors of the aqueous flow) seems incapable of preventing the pathogenous evolution of this type of glaucoma[1, 2] and, what is even worse,[3–4] by lowering the tension without opening the angle, this conservative treatment may give a false sense of security. If this phenomenon, which at the beginning is only a functional unbalance, favored by an anatomic characteristic (narrow-angle), persists, it can be transformed into a definite organic defect (goniosynechias). Medical treatment is, therefore, justified only as preliminary to or complementary to surgical treatment.

A different procedure is indicated for the open-angle glaucoma—medical treatment should be chosen first, the surgical treatment being given only when medical therapy (miotics or inhibitors of aqueous flow) is incapable of controlling the tension or the progressive loss of visual fields. Our experience agrees with that of Scheie, et al.,[5] that, up to

the present, in this type of glaucoma, repeated tonometric controls and frequent perimetric examinations are more important than normalization of the coefficient of outflow (C). It is possible that in the future wider tonographic experience will permit us to reach more exact conclusions in this respect, as is suggested by other authors of well-known prestige.

From a clinical point of view and to effect the classification of different cases for their interpretations and to guide therapy better, we prefer to speak of open-angle glaucoma or closed-angle glaucoma. It is evident that the latter is the result of the narrow angle and, for it, this term would be enough but, as there are open-angle glaucomas that occur in patients with narrow angles (narrow open-angle), it is not possible to accept a classification which might lead to confusion and a wrong interpretation of the pathogenesis.

Apart from the clinical symptoms that may guide us toward one or the other pathogenic form, it is necessary, in order to classify a given case, to do a gonioscopic examination during the hypertensive episode, using in doubtful cases certain useful techniques—observation of the paralactic shift of the narrow beam,[6-8] or the off-on test.

According to the tonometric, tonographic, and gonioscopic criteria (for the latter we used the Shaffer graph[10]), we have divided and classified the different forms of glaucoma for clinical and therapeutic guidance[11-a, b] (table 1). Previously we have described the details of our classification.

Fundamentals of surgical treatment

In our opinion,[4] three important principles must rule the choice of surgical procedure for the primary glaucoma: (1) Perform surgical procedures according to the pathogenesis of the case; (2) avoid unnecessary major surgery which, instead of resolving the problem definitely, might worsen it; (3) recognize a degree of importance for the different surgical treatments, according to the cases' state of evolution (this is particularly necessary in mechanical glaucomas [narrow closed-angle]).

TABLE 1

Clinical and pathogenic interpretation

A. Narrow angle	1. Closed angle	a. Preglaucoma	Normal tension and outflow (C over 0.12) when the angle is open
		b. Interval closed-angle	
		c. Chronic closed-angle	Abnormal tension and outflow (C under 0.12) with definite closed angle by anterior synechias
	2. Closed-open-angle (closed & open angle mixed pathogenesis)	A. Primary closed-angle & secondary open-angle due to trabecular damage by repeated contacts.	Abnormal tension and outflow (C under 0.12) after the attack when the angle is open
		B. Primary coexistence of both mechanisms?	
	3. Open-angle	Mechanical pathogenesis is not present at the time but is predisposed by the anatomy configuration. Generally Shaffer's type angle 1 or 2.	
B. Wide angle	Open-angle	Shaffer's type angle 3 or 4.	

1. Pathogenesis

Grant,[12] Goldmann,[13] and Weekers and Prijot[14] have clearly demonstrated that high ocular tension is due in most cases to an impairment of the aqueous outflow. It is our opinion, however, that such "flow" reducing procedures as cyclodiathermy, cycloelectrolysis, angiodiathermy, and so forth, are not pathogenic operations, and therefore should not be procedures of first choice for primary glaucoma, except in the very few cases of hypersecretion glaucoma (two percent[15]), with which we have had no experience; or eventually in those patients with greatly reduced visual fields and not very high tension, where a major operation might be dangerous. We believe that, except in these two instances, one probable, the other certain, such operations should be performed only when the desired results have not been obtained with another type of operation.

In closed-angle glaucoma, considering the mechanical etiology of the hypertension, peripheral iridectomy or eventually an iridotomy—the really pathogenic methods—are the only ones which will arrest, as Barkan[16] states, the progressive iris bombé, the relative pupillary block, and the gradual narrowing of the chamber and the angle. By this technique, if evacuation of the anterior chamber is avoided, new blockages of the iris against the trabecula and the ever-frightening possibility of definite damage or the formation of an anterior synechias are prevented.

Peripheral iridectomy can restore the "physiologic conditions," as stated by Barkan, resulting in normal tension and outflow, only if the trabecula is definitely freed from the iris and if it is sufficiently permeable to permit drainage of aqueous. That is to say—this operation can only fulfil its healing purposes in simple or not complicated cases of closed-angle glaucoma (subgroups a and b of our classification[11-a, b]) because they are not associated with definite synechias or are not complicated by some other mechanisms.

This clearly shows the great importance of correct clinical interpretation which, in our opinion, can actually be obtained by gonioscopy, tonometry, and tonography. (Tonography is essential for the classification.)

With a suitable surgical technique, the peripheral iridectomy is a reasonably safe operation and, for this reason, it is indicated not only as a means of healing but also as a prophylactic measure for preglaucoma cases, especially when one considers that the continuous use of miotics or inhibitors of aqueous flow do not prevent the development of the closed-angle glaucoma.

In the chronic cases of closed-angle glaucoma (subgroup c) or in those with a mixed pathogenesis (group 2), peripheral iridectomy seems not to have the real healing value as in the simple forms previously mentioned; it can only, at best, mitigate the recurrence of acute attacks by deepening the chamber. Any outflow impairment which is present is going to persist and the surgical treatment will then be incomplete and is only justified in those cases of mixed pathogenesis, in which the intraocular pressure is controlled by the use of miotics when the angle is open, and the facility of outflow is over 0.12 mm.^{3}/min./mm. Hg.

For these cases Barkan recommends the peripheral iridectomy to deepen the chamber and, if the tension is not modified afterward by the medical treatment, he resorts to other types of surgery. He prefers cyclodialysis because he considers it less exposed to complications than the classical filtering operations, which, according to his point of view, predispose to or aggravate the mechanical blockage of the angle by development of peripheral synechias. Although some authors perform the classical filtering operations in these cases, the majority prefer iridencleisis. We agree with Barkan's criteria especially because, in our opinion, they respect the

principles that must control the surgery of the glaucoma.

In order to make it unnecessary to submit patients with this type of glaucoma to more than a single intervention, we have devised an operation which we call "filtering iridectomy," and with which we have obtained promising results.[11-b] By this technique, a deeper anterior chamber is obtained. Equalizing the tension on both sides of the iris will prevent new blockage and, at the same time, by the addition of diathermic coagulation on both sides of the incision, a permanent filtration is obtained to normalize the tension and the outflow of aqueous that cannot be obtained through classic peripheral iridectomy. We prefer this operation to the classic filtering techniques for the following reasons:[4] (a) It is less traumatic; (b) it is possible to avoid evacuating the chamber; (c) it modifies the mechanical component of the hypertension in the same way that the simple iridectomy does; (d) generally a round pupil is obtained; (e) the anatomy is better preserved than in other external filtering operations which in a high percentage of cases, favor the formation of goniosynechias, leaving the entire success of the operation to formation of a filtering bleb. With our technique the trabecular area is freed of proximity to the iris and thus permits outflow of aqueous, if the trabeculae are in good condition, in such a way that the result does not depend solely on the filtering scars.

In narrow open-angle glaucoma (chronic simple glaucoma, group 3), the filtering iridectomy is also preferred, as the anatomic configuration that predisposes to the development of such a mechanical type of glaucoma is able to hasten its evolution when other types of surgery are used.

In wide open-angle glaucoma the classical filtering operations are more efficient and there are fewer chances of postoperative complications of the malignant glaucoma type. Logically the filtering iridectomy is also recommended in these cases and, in the few cases in which we have used it, the results obtained were satisfactory.

Until now we have attempted to treat surgically this form of primary simple glaucoma (wide open-angle) with the superior and inverse cyclodialysis and the results on the ocular tension were very good. The surgical cleft is more constant and more frequently observed with this technique than with the ones we have tried previously. A larger or smaller cleft corresponds to a more or less marked lowering of the intraocular pressure postoperatively. However, as the cyclodialysis is a more difficult operation to control,[17] since it carries the risk of unforeseen complications in spite of a perfect technique, and because it seems that its action is not clearly understood, as well as because it appears not to be the technique pathogenically best adapted to the source of the hypertension in wide open-angle glaucoma, at present we do not consider this procedure a primary choice for this type of glaucoma.

At present we are using the following criteria for the surgical indication of the different clinical forms of glaucoma:

a. Preglaucoma		Peripheral iridectomy
b. Interval closed-angle		
c. Chronic closed-angle*		
d. Mixed type	primary, secondary	Filtering iridectomy
e. Narrow	open-angle	Filtering iridectomy
f. Wide	open-angle	Filtering iridectomy or any other classical filtering operation (Elliot, Lagrange or iridencleisis

* We regard cyclodialysis as a second choice for subgroups c, d, e, and f.

We consider it unnecessary to give the technical description of the procedure for the peripheral iridectomy, as, in general, it follows Barkan's directions. Also we are not mentioning the results obtained with it, except they are in accordance with the majority of authors, as we have already commented.[11-b] The same applies for the cyclodialysis.[11-b]

We believe that the description of the "filtering iridectomy" technique might be

more interesting, as well as comments on the results obtained.

Filtering iridectomy: Surgical technique*

1. One drop of two-percent eserine three hours before the operation.

2. If the patient has undergone a congestive attack during the preoperative period, we usually prescribe acetazolamide (250 mg.), three times a day, and a daily intramuscular ampule of Irgapyrine.

Operation

1. Subconjunctival injection of two-percent Xylocaine which helps to form a good cleavage in the conjunctival flap, which is best conserved when infiltrated.

2. Oblique puncture of the chamber with a piece of razor blade or a keratome at the 3-o'clock or 9-o'clock position to avoid evacuation and to prepare a channel in case it is necessary to deepen the chamber at the end of the operation; thus dangerous maneuvers are avoided.

3. Conjunctival incision, 12 mm. to 13 mm. from the corneal limbus.

4. Dissection of a conjunctival flap with a keratome or piece of razor blade mounted on a needle holder.

5. After coagulation of the superficial vessels with a strabismus hook, a 1.5- to 2.0-mm. incision is made with a piece of razor blade (ab externo) behind and parallel to the limbus through the superficial two thirds of the scleral lamellae and lengthened to 4.0 to 5.0 mm.

6. Both lips of the scleral incision are touched with a thin diathermic needle and its aperture is observed. This operation is repeated on the internal edge of both lips to enlarge the cleft further. Three arc-shaped rows of surface diathermy with a forward-directed concavity are added, with two mm. distance between each of them, so that a distant cicatricial retraction is obtained and, in this way, the cleft is further enlarged.

7. With a piece of razor blade directed upward, the remaining one third of the scleral incision is completed. Generally by this time the iris spontaneously prolapses but, if it does not, both edges of the incision are pressed with a blunt instrument, this being sufficient to obtain the desired results. Actually it is never necessary to introduce forceps into the anterior chamber.

8. The iridectomy is performed with a curved scissors. Care must be taken not to touch the wound edges, thus preventing evacuation of the chamber.

9. The conjunctival flap is folded back and, using a cotton swab, the limbus region is massaged so that the pupil is rounded. In some instances we use a drop of acetylcholine subconjunctivally.

10. Then the conjunctival incision is closed with separate stitches, using virgin silk.

11. If the anterior chamber is flattened or completely emptied, air is injected into it through the previously made incision (see step 2).

12. Binocular bandages are applied for 24 hours.

Postoperative care

Remove the bandage of the healthy eye after 24 hours. The operated eye is left covered for four or five days. Hydrocortisone with neomycin is instilled daily and a weak mydriatic is used after three or four days.

In spite of the extreme care taken to avoid the loss of the anterior chamber during the postoperative period, this has happened in four cases. A marked iris atrophy after an acute attack was observed in three of these cases, and this is the reason why we will comment on these cases later. In such cases it is better not to insist on the miotic therapy for more than three or four days but, if the

* We have learned recently through Derrick Vail's comments at the 63nd annual session of the American Academy of Ophthalmology and Otolaryngology (Am. J. Ophth. **44**:836-840, 1957) that independently from us Dr. Harold Scheie has reported on a similar technique (Retraction of scleral wound edges as a fistulizing procedure for glaucoma).

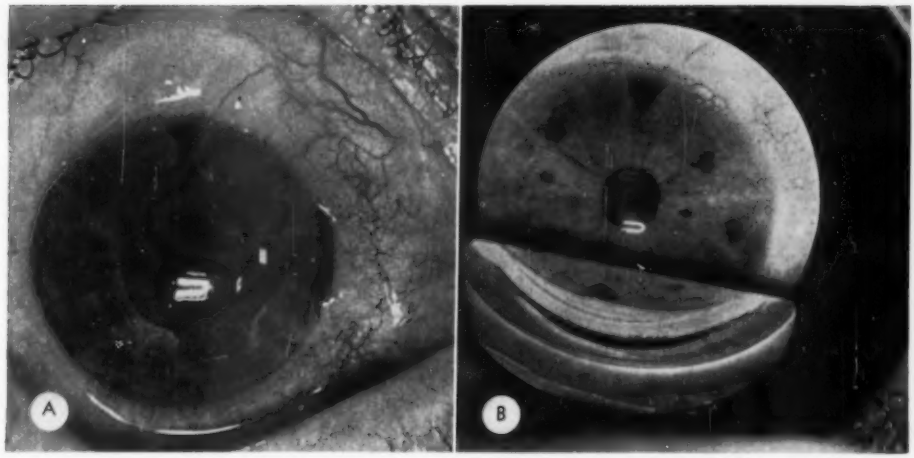

Fig. 1 (Malbran and Malbran). (A) Postoperative gross appearance. Note the filtering bleb. (B) Postoperative gonioscopic view.

abnormality persists, it is necessary to prescribe a weak mydriatic. This is enough to deepen the chamber. It would seem that the mydriasis would interrupt a probable pupillary block.

The filtering bleb may be present from the very first days after operation, but sometimes it is not evident until 20 or 30 days later and, by this time the tension is normalized. This possibility must be taken into consideration before risking a new operation; to be on the safe side a miotic and acetazolamide are used.

If the pupil is not round by the end of the operation, it very seldom will be, although sometimes the inclusion of an iris pillar in the wound serves as an iridencleisis, thus solving the situation. Some of our failures were due to this and, upon gonioscopic examination, we could observe the adherence of the iris to the cornea, with trapezoidal-shaped synechias by which the iridectomy was obstructed. Because of this the operation failed in its double purpose of deepening the chamber and obtaining a filtering scar.

Statistical comments (table 2)

1. Good results (41 cases)

Normalization of the tension (under 23.4 mm. Hg) (Schiøtz-Mueller electronic tonometer) is considered as a good result as well

TABLE 2

Results in 50 cases

Number of Cases	Type of Glaucoma	Good Results	Borderline Results	Failures
	Chronic closed-angle (21 cases)	16	3	2
	Mixed type (14 cases)	12	1	1
	Narrow open-angle (12 cases)	10	2	0
	Wide open-angle* (3 cases)	3	—	—
50		41 (82%)	6 (12%)	3 (6%)

* We recently adopted this technique (filtering iridectomy) for wide open-angle glaucoma. This is the reason for the small number of cases reported in these statistics.

as an evident improvement of the coefficient of outflow when compared with the presurgical status. Pre-and postoperative tonography was done in 33 cases. The average coefficient obtained ranged between 0.22 mm.³/min./mm. Hg, with a minimum of 0.12 (one case) and a maximum of 0.40 (one case).

In 17 cases tonographic studies were not possible; 12 cases were in one-eyed patients and in five cases we did not have sufficient cooperation. Included in the 41 cases in which good results were obtained is one patient with a chronic closed-angle and marked atrophy of the iris after an acute attack. In this case tension improved to 10 mm. Hg., with an outflow of 0.30 (with good filtering bleb) but, as the chamber became flat during the postoperative period (third day) and as no other treatment (miotics, mydriatics, and two air injections) was successful, it was necessary to remove the lens which had undergone cataractous changes.

In five cases, additional miotic therapy was necessary. In three of them with flattened scars, miotics were necessary immediately postoperatively. In the two remaining cases miotics were necessary after eight months and one year, respectively. Reduction of the initial bleb was also observed in these two cases.

Of the successful 16 cases of chronic closed-angle glaucoma, five were postacute. In two of them, showing atrophy of the iris, the anterior chamber became flat during the immediate postoperative period. One improved on medical therapy and the other, previously mentioned, was the one in which extraction of the lens was necessary.

2. Borderline results (six cases)

The borderline results were in those cases in which the tension improvement was evident but in which the postoperative tension ranged between 25 mm. and 29.6 mm. Hg in spite of miotic therapy.

3. Failures (three cases)

In two cases the preoperative state was not modified. The one remaining case developed malignant glaucoma with loss of the anterior chamber and lens extraction was necessary.

Complications

Flat chamber (four cases). All of these cases occurred during the immediate postoperative period. In three cases there were nuclear cataracts with marked postacute attacks of atrophy of the iris. It is possible that, if the iris fails as a retention diaphragm, the forward shifting of a swollen lens is favored. Two cases were improved on miotic therapy; in the other two, total extraction of the lens was necessary.

Distorted pupil (nine cases). In five of these cases good results were obtained and the small portion of the iris which was included in the wound played the part of an iridencleisis. Of the remaining four cases two are classified in the borderline group and the other two as failures.

In the nine cases in which the pupils became distorted, goniosynechias, which were not present preoperatively, were observed. This is a point that must be remembered. In our experience when the pupil is rounded at the time of the intervention and the chamber is formed, new goniosynechias are seldom added to the old ones present preoperatively.

Time elapsed since surgery

Follow-up in the cases presented in these statistics ranged between two and 22 months.

Summary and Conclusions

We have to stress the surgical character of this treatment for mechanical glaucoma (narrow closed-angle) and also the medical character of the treatment for open-angle glaucomas, in which surgery is only a complementary method when the tension is not normalized and there is progressive loss of visual fields.

We prefer to use the terms "closed-angle" and "open-angle" better to describe the path-

ogenesis of a given case and better to decide on the therapy to be followed.

The proposed classification bases the different types of glaucoma upon gonioscopic, tonometric, and tonographic criteria.

Useful surgical methods for each type of glaucoma in this classification are mentioned and a new surgical technique is described for peripheral iridectomy which is particularly useful in cases with a mechanical pathogenesis (narrow closed-angle) or mixed (narrow closed- and open-angle) in which tonography and gonioscopy have shown that there is an uncontrollable impairment of the outflow in spite of medical treatment and in which the usual peripheral iridectomy is not a sufficient therapeutic measure.

An analysis is made of the good results obtained with this new technique. It is also proposed as a procedure in cases in which there is no actual mechanical pathogenesis (narrow open-angle and wide open-angle).

Parera 94.

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BILATERAL HOMONYMOUS HEMIANOPSIA*

Clinics in Perimetry, no. 2

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Bilateral homonymous defects in the visual fields are unusual. The majority are due to occlusive vascular disease resulting in infarction of the occipital lobes, although rarely a single meningioma of the falx cerebri may compress both occipital lobes and also give rise to bilateral defects.

The homonymous defects may appear first on one side and then on the other or they may appear simultaneously in both half fields. The sudden and simultaneous development of bilateral homonymous field defects and signs of a lesion in the brain stem, such as nystagmus or internuclear ophthalmoplegia, appear to be diagnostic of occlusive disease of the basilar arterial system.

We have encountered an increased number of bilateral homonymous field defects due to vascular disease over the last five years at the Mayo Clinic. Whether this increase is actual or merely reflects the increased awareness and interest in occlusive disease of the basilar arterial system is difficult to say.

Although no sharp demarcation into types can be made, three general varieties are apparent. The first is an almost complete loss of peripheral field with preservation of only a small island of vision near the point of fixation. The second is bilateral homonymous scotomas with normal peripheral vision. The third is bilateral homonymous quadrantal hemianopsia. The patient with the first type suffers the most intense handicap and may appear almost blind except for perception of movement and light. As time goes on, even though the fields show little improvement, the patient seems to use his remaining field with better facility and may even be able to read, though with difficulty. The patient with bilateral homonymous scotomas also is handicapped severely and is usually unable to read, since he can see only a few letters at a time and cannot tie them together. Bilateral homonymous quadrantal defects cause the least handicap. The defects may be of the crossed variety with the upper fields of one side affected and the lower fields of the other side affected; they may involve the upper fields of both sides or, more commonly, both lower fields. Why the bilateral homonymous quadrantal defects are so frequently inferior, producing an inferior altitudinal anopsia, to our knowledge, has not been satisfactorily explained.

An example of severe loss in both the right and left homonymous fields with small preserved central isles of vision is supplied by the following case.

Report of cases

Case 1.

A man, 59 years of age, complained of difficulty in seeing and in walking. Three months previously his relatives had noted that his speech was slurred and that he scuffled when walked. A week later the difficulty with talking and walking increased and vision to the right side was lost. Two weeks before admission to the clinic he suffered a "stroke" characterized by rolling and thrashing about in bed and followed by further loss of vision and numbness of the right side of the face and in the right arm.

The ocular fundi appeared normal. Visual fields (fig. 1) showed intense contraction with residual central isles of vision, the consequence of right and left homonymous hemianopsia with a small amount of preservation centrally, attributed to infarction in both occipital lobes. In addition, examination of the eyes disclosed loss of sensation on the cornea of each eye, mild weakness of the right orbicularis oculi, moderate paresis of conjugate gaze to the right, and internuclear ophthalmoplegia on attempted gaze to the left. These signs pointed to involvement of the brain stem, chiefly of the pons. All of the affected areas are supplied by branches of the basilar artery.

* From the Section of Ophthalmology, the Mayo Clinic and the Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

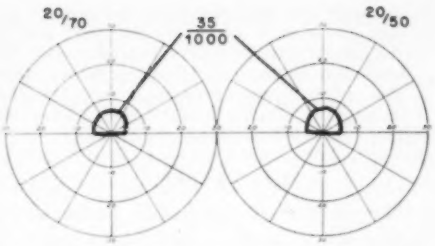

Fig. 1 (Rucker and Kearns). Residual central isles of vision.

The following case provides an example of bilateral homonymous scotomas.

CASE 2.

A 71-year-old woman who was seen at the clinic in April, 1957, complained of numbness of the left hand and difficulty with her vision. The onset was sudden, on the afternoon of April 19, 1957. The numbness of her hand had disappeared by the next morning but her husband noted that she did not see things on her left when he offered them to her. Visual fields, plotted three days after the onset of her difficulty, showed complete left homonymous hemianopsia dense to even the largest targets.

By the following day her visual fields were improving. The neurologist made a diagnosis of cerebral infarction in the distribution of the right posterior cerebral artery. Anticoagulant therapy was discussed but deferred in view of her spontaneous improvement. After she returned home, her vision improved further, and became what she considered normal.

She continued well until the morning of January 10, 1958. She arose as usual and felt well but while pouring orange juice into a glass at the breakfast table she found that she could not see clearly. She later found that she was unable to read and, although she could write, it was difficult because she could not see what or where she was writing.

On re-examination at the clinic she could name letters but could not read. Her peripheral fields were grossly full, which raised the possibility that the reading difficulty might be a visual agnosia. However, visual fields were charted (fig. 2) and disclosed bilateral homonymous scotomas, a loss great enough to be a handicap in seeing to read. The neurologist interpreted these fields as evidence of bilateral cerebral infarction of the occipital lobes, probably as a result of basilar artery insufficiency.

The bilateral quadrantal defects may involve only the lower halves of the fields as in the following case.

CASE 3.

A 57-year-old man came to the clinic in November, 1956, for evaluation of his diabetes and for relief of residuum of a cerebral vascular accident

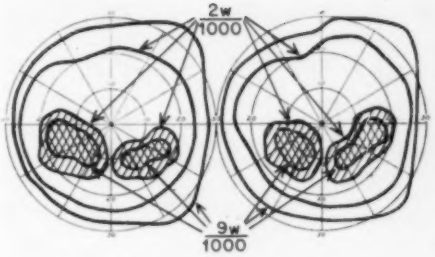

Fig. 2 (Rucker and Kearns). Bilateral homonymous scotomas.

suffered four years previously. He gave a history of sudden onset of dizziness, paralysis of the left arm and leg, numbness of the left side of the body, and visual difficulty. He found that in attempting to read, he had to move the page of print or turn both his head and eyes to see it well. He gradually regained the use of his extremities but the visual difficulty persisted.

On examination he was found to have mild diabetic retinopathy consisting of scattered microaneurysms in each eye. The central vision was 20/20 in each eye but there was a gross loss of both lower visual fields. Visual fields were plotted and, as can be seen in Figure 3, there was a bilateral homonymous defect producing an inferior altitudinal defect in each eye.

Neurologic examination showed ataxia and diminished corneal sensation on the left. It was the opinion of the consulting neurologist that the bilateral homonymous field defects with these signs of involvement of the brain stem indicated infarction in the distribution of the basilar system.

Sometimes the quadrantal defects appear in both the right and left halves of the fields, and may be greater on one side than the other. An example of right and left homonymous hemianopsia of simultaneous onset follows.

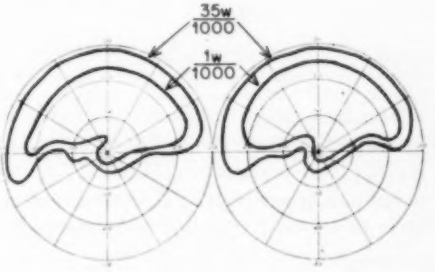

Fig. 3 (Rucker and Kearns). Inferior altitudinal anopsia.

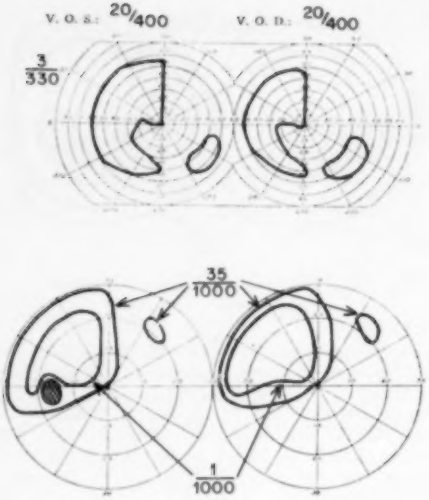

Fig. 4 (Rucker and Kearns). Bilateral homonymous hemianopsia.

CASE 4.

A 56-year-old man came to the clinic on March 3, 1958, for advice regarding visual loss. Four months previously he had become weak and confused and was taken to a hospital. After a few days he fell into a coma which lasted a day or two, and after a week of confusion he gradually improved. At that time he found he was nearly blind, but shortly regained a portion of his sight. On admission to the hospital it was found that he had suffered a severe hemorrhage from a duodenal ulcer. Eight transfusions were administered.

On admission to the Mayo Clinic the visual disturbance was explained by the finding of bilateral homonymous hemianopsia (fig. 4). The ocular fundi showed mild narrowing and sclerosis of the retinal arterioles, hypertensive type. Neurologic examination did not yield abnormal findings. The consultant in neurology concluded that there had been an impending occlusion or stenosis of the upper portion of the basilar artery at its bifurcation into the posterior cerebrals and that the hypotensive effect of the intestinal hemorrhage was the final blow which produced infarcts in both occipital lobes. He regarded further improvement as unlikely.

SUMMARY

The perimetrist occasionally encounters a patient who presents both right and left homonymous hemianopsia. In some instances the bilateral loss may be attributed to two distinct episodes separated by an interval of some time; in that case the lesions may be two separate vascular accidents involving both optic radiations or the visual cortex of both occipital lobes. In other instances both sides of the visual fields suffer losses simultaneously; in that case the offending lesion is due to occlusive disease in the basilar artery and its branches. That the disease is in the basilar artery is supported by the frequent association of other neurologic disorders arising in the pons, a structure also supplied by this vessel. Pontine involvement is suggested by signs about the eyes which include nystagmus, internuclear ophthalmoplegia, abducens paralysis, weakness of the orbicularis muscles, corneal or facial hypesthesia. The neurologist may find other supporting evidence, such as impairment of the motor and sensory projection systems, cerebellar pathways, and cranial nerve nuclei.

The Mayo Clinic.

METASTATIC CARCINOMA TO THE RETINA*

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The metastasis of a tumor to the eye is not an infrequent occurrence. The uveal tract is the tissue of predilection. Maxwell[1] in 1954 noted that approximately 275 cases of metastases to the uveal tract had been reported. In the majority of these reports the choroid had been affected. Involvement of the anterior uveal tract is much less frequent, fewer than 40 cases having been reported.

The metastasis of a tumor directly to the retina is an event of the utmost rarity. We have been able to find one reported case. Smoleroff and Agatson[2] in 1934 reported the

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metastasis of a gastroesophageal carcinoma to the retina without involvement of any other portion of the globe.

We have recently observed a case of metastatic carcinoma of the retina. The infrequency of this type of metastasis, and the unusual problems of diagnosis and treatment encountered in our case, lead us to report it.

Case history

The patient, (E. D., JHH #576101), a 60-year-old white woman, was first seen by one of us (F. B. W.) on May 24, 1951, with the complaint of decreased vision in the right eye.

At age three years she had had convulsions after which the vision in the left eye had never been good. Since the age of nine years she had worn glasses for myopia. During the past year she had noted gradual, painless loss of vision in the right eye. On examination, the best corrected vision in the right eye, with a —9.0D. sph. was 20/100; in the left eye with a —7.0D. sph. it was 6/100.

Ophthalmoscopic examination of the right eye revealed lens opacities, floating vitreous opacities, a normal disc, patches of old chorioretinitis to the nasal side of the disc, and a normal macula. In the left eye the lens changes were more pronounced and the macula was not seen. One patch of chorioretinal atrophy was seen above the disc, and another in the temporal fundus. Because of the long history of defective vision in the left eye, removal of cataract from the right eye only was advised.

On May 26, 1951, an entirely uneventful intracapsular cataract extraction with peripheral iridotomy on the right eye was performed. The postoperative course was uneventful, and she obtained, with correction, a visual acuity of 20/30 in the right eye.

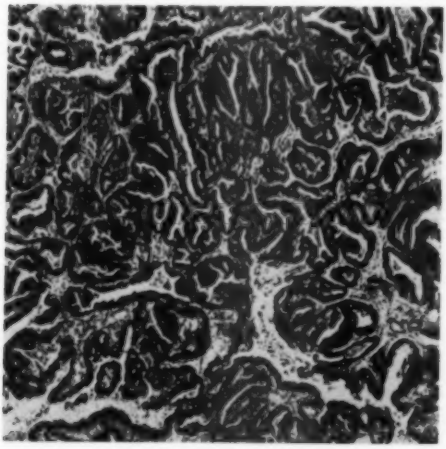

Fig. 1 (Duke and Walsh). Adenocarcinoma of fundus of uterus, showing moderately well-differentiated glandular acini (hematoxylin-eosin, ×75).

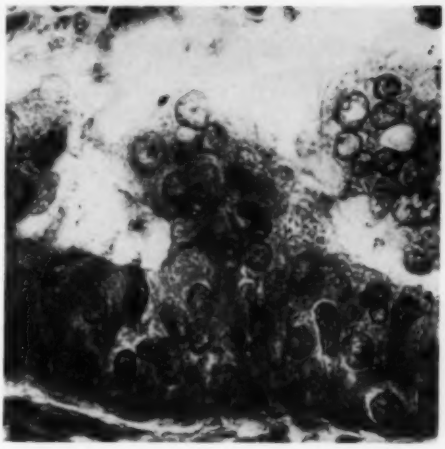

Fig. 2 (Duke and Walsh). Morphology of cells of uterine adenocarcinoma (hematoxylin-eosin ×725).

On April 18, 1952, she returned to The Johns Hopkins Hospital with the chief complaint of painless bleeding from the vagina of three days' duration. On April 22, 1952, under general anesthesia dilatation and curettage were performed. There was a large amount of white, friable tissue in the uterine cavity. Radium, 75 mg., was inserted in the uterine cavity and removed two days later. Microscopic examination of the curettaged material showed an atrophic cervix and adenocarcinoma of the uterine fundus, Grade III (figs. 1 and 2).

On June 2, 1952, she was admitted to The Johns Hopkins Hospital for the third time and on the following day a total hysterectomy, bilateral salpingectomy, and left oophorectomy were performed. (The right ovary had been removed 30 years previously.) She was discharged from the hospital on June 15, 1952.

On July 12, 1954, the patient returned with the complaint of sudden failure of vision in the right eye of one month's duration. The best corrected vision in the right eye was 5/100 and ophthalmoscopic examination revealed a small disc-sized, white area of elevation directly at the fovea. Areas of old chorioretinitis were seen in the far periphery in all quadrants. There was a central scotoma. The ocular tension was 16 mm. Hg (Schiøtz). Examination of the left eye revealed a mature cataract. Fundus details could not be seen. It was felt that the central lesion in the right eye probably represented a Junius-Kuhnt disciform degeneration. Cataract extraction, left, eye, was suggested, but the patient returned home without treatment.

On September 2, 1954, she entered The Johns

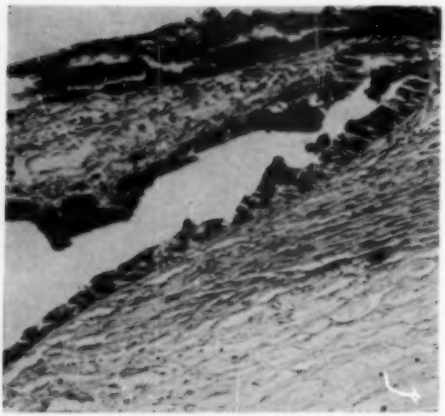

Fig. 3 (Duke and Walsh). Tumor cells lining the posterior surface of the cornea, the chamber angle and the surfaces of the iris (hematoxylin-eosin ×125).

Hopkins Hospital for the fourth time. The right eye had been red and irritated for three weeks. Examination disclosed an ocular tension of 40 mm. Hg (Schiøtz), corneal edema, and a few keratic deposits on the back of the cornea. The macular lesion was unchanged. She was placed on miotics. A gynecologic consultation revealed no evidence of any recurrence of the uterine carcinoma removed 27 months previously.

On September 3, 1954, intracapsular cataract extraction with peripheral iridotomy, left eye, was performed.

The elevated ocular tension of the right eye failed to respond to miotics and Diamox, and on September 13, 1954, a cyclodiathermy and paracentesis were performed, followed on September 21st by a cyclodialysis. These procedures failed to control the tension and on September 24, 1954, the right eye was enucleated (EP #15004).

The findings on microscopic examination of the eye are reported in detail. The basal cells of the corneal epithelium showed a moderate degree of swelling and edema. At the limbus on one side there was a through and through surgical scar. Also here there was a peripheral iridectomy. The anterior chamber, of average depth, was filled with a moderate amount of fibrin and a few red blood cells. The peripheral aspects of the posterior surface of the cornea, the chamber angle, and the anterior surface of the iris (figs. 3 and 4) were lined by a continuous and uninterrupted layer of neoplastic epithelial cells, two or three cells in thickness. These cells were of moderate size, had a moderate amount of eosinophilic staining cytoplasm, and prominent round or oval basophilic nuclei. Occasional cells were vesiculated. Mitotic figures were numerous and there was a moderate degree of pleomorphism in cell type. However, these cells, cuboidal or columnar in shape, had a definite tendency to arrange themselves rather uniformly, with a suggestion of vertical polarity to the base on which they were lying. The iris itself was thinned and atrophic with some fresh hemorrhage within the stroma. In the limb of the iris in which the iridectomy had been performed there was considerable necrosis with disruption of the pigment epithelium and liberation of pigment within the stroma of the iris. The ciliary body was markedly atrophic with hyalinization of the ciliary processes. Extensive chorioretinal scars were noted in the far periphery extending posteriorly almost to the equator. The retina in these regions had been practically destroyed having been replaced with numerous small cystoid spaces and a considerable proliferation of glial tissue. The neoplastic cells which lined the anterior surface of the iris continued over the pupillary margin and appeared to line the posterior surface of the iris. They extended in a sheet over the ciliary processes and back along the surface of the pars plana. Posteriorly, in a practically continuous sheet, they lined the internal surface of the retina sending small branches out into the vitreous (fig. 5). The cells covered the optic disc as well (fig. 6). In the macula and perimacular area large clumps and nests of neoplastic cells were seen lying within the retina itself (figs. 7 and 8). These cell nests were predominantly in the nerve-fiber and inner-plexiform layers. Several of them completely surrounded blood vessels. There had been considerable disturbance of the architecture of the retina here with extensive hemorrhage, necrosis, and polymorphonuclear leukocyte infiltration into the innermost layers of the retina. There was also degeneration and considerable loss of the rods and cones in the region between the macula and the disc. Medial to the optic disc were several old atrophic chorioretinal scars. The optic nerve had been severed flush with the sclera. It is worth emphasizing that no tumor cells were found within the choroid or within the stroma of the ciliary body or iris.

Microscopic slides of the uterine curettings and of the uterus itself were reviewed. The cell type of the metastatic tumor tissue within the eye corresponded closely to the original uterine adenocarcinoma.

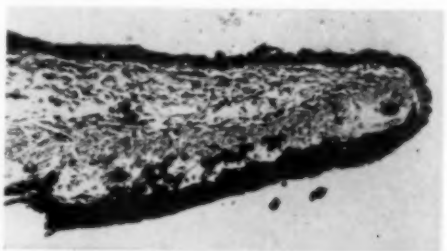

Fig. 4 (Duke and Walsh). Extension of tumor cells around pupillary margin of the iris (hematoxylin-eosin, ×125).

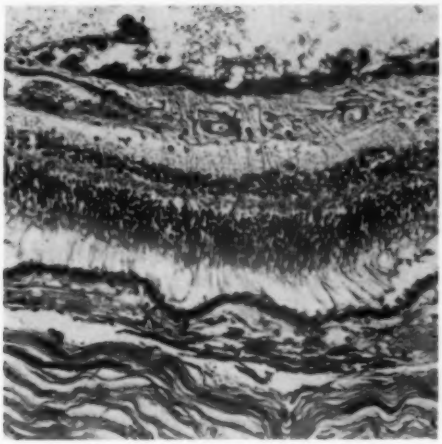

Fig. 5 (Duke and Walsh). Extension of tumor cells on inner surface of the retina (hematoxylin-eosin, ×125).

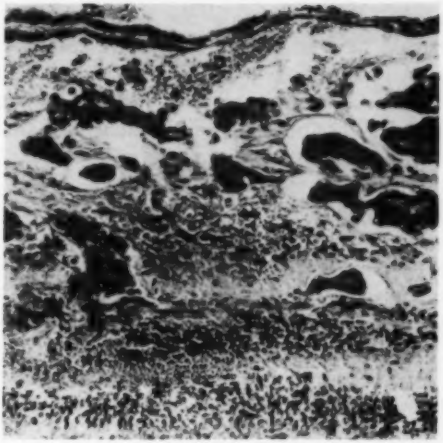

Fig. 7 (Duke and Walsh). Retina, perimacular area. Large nests of tumor cells lying predominantly in nerve fiber and ganglion cell layers (hematoxylin-eosin, ×125).

The final pathologic diagnoses were adenocarcinoma, uterine, metastatic to retina with extension to ciliary body, iris, and cornea; secondary glaucoma; chorioretinitis, atrophic; aphakia, surgical.

The exact details of the patient's course following enucleation are not known. However, early in 1955 she developed excessive thirst, which was controlled with pitressin. A few weeks later she developed symptoms of cardiac failure and died. The development of diabetes insipidus suggests the possibility of metastasis to the brain. Autopsy was not obtained. The patient survived approximately three years after the removal of the primary uterine carcinoma and for a few months after removal of the eye with the metastatic tumor.

Comment

The clinical manifestations of this metastatic tumor to the eye were unusual. The ocular complications occurred two years after removal of the primary uterine carcinoma. The neoplastic cells, blood borne, ap-

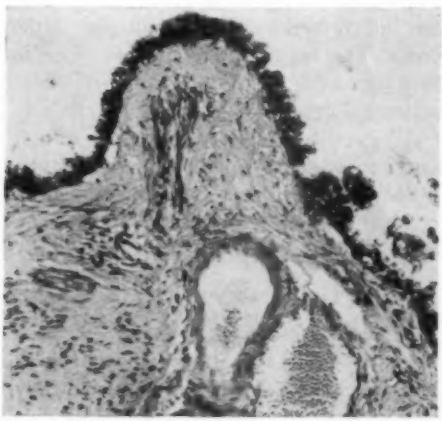

Fig. 6 (Duke and Walsh). Extension of tumor cells over surface of optic disc (hematoxylin-eosin, ×125).

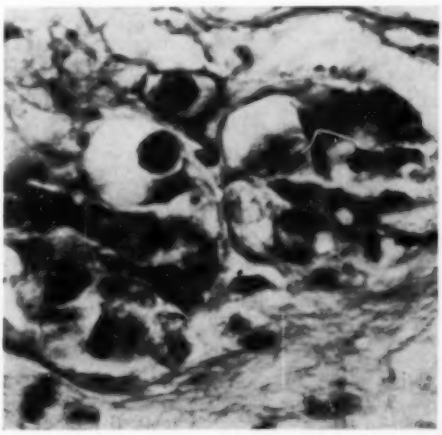

Fig. 8 (Duke and Walsh). Morphology of metastatic tumor cells in the retina (hematoxylin and eosin, ×725).

parently lodged within the retina in the macular region. Proliferation there resulted in marked reduction of visual acuity. This mass of tissue in the macular area was interpreted clinically as a Junius-Kuhnt type of disciform degeneration. The neoplastic cells at this site ultimately broke through the internal limiting membrane of the retina and extended along its inner surface in all directions, covering the disc as well. Tumor cells extended anteriorly and covered the surface of the ciliary body, iris, and chamber angle. Occlusion of the filtration angle by neoplastic cells produced intractable glaucoma. Clinically, the glaucoma was thought possibly to have been related to the uncomplicated cataract extraction performed three years previously. After all measures to control the glaucoma had failed, the eye was enucleated. Microscopic examination revealed the unique course of events.

Summary

A case of metastatic carcinoma to the retina, the second such case in the literature, has been reported. The unusual clinical aspects of the case and the findings on pathologic examination have been described.

The Johns Hopkins Hospital (5).

Addendum

Since this paper was submitted for publication, Kennedy, et al. (Arch. Ophth., **60**:12-18 (July) 1958) have reported a patient with metastasis of a carcinoma of the rectosigmoid to the macula of the right eye.

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CORNEAL TRANSPLANTS AND BLOOD TYPES*

A clinical study

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An immune response has been offered as the explanation for the late clouding of some technically successful corneal grafts. Most authors have employed animals, usually rabbits, to study this problem.[1-5]

The ubiquity of ABO antigens in the plant and animal world has long been known.[6,7] Nelken, et al.,[8,9] have demonstrated the presence of ABO antigens in the human cornea and ABO antibodies in the aqueous humor, but could not detect Rh antigens in the cornea. The reported detection of rises in the serum antibody titers in some ABO incompatible grafts by Klen[11,12] and Nelken, et al.,[9,10] posed the question of the role of these antigens in the opacification of grafts. In the present study an attempt was made to correlate donor-recipient blood type incompatibility with the results of corneal grafts.

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Case material

The study was based on 68 corneal transplants performed by resident and staff members of the Department of Ophthalmology, Washington University School of Medicine. Blood from the donors and recipients of the grafts was typed. All transplants were in-

TABLE 1

ABO COMPATIBILITY

	Total	Compatible	Incompatible
No. grafts	68	50	18
Successes	22	15	7
Failures	46	35	11
% successful	32	30	39

TABLE 2

ABO AND RH COMPATIBILITY

	Total	Compatible*	Incompatible†
No. grafts	63	40	23
Successes	21	12	9
Failures	42	28	14
% successful	33	30	39

* ABO and Rh compatible.
† ABO or Rh incompatible.

cluded in this series in which adequate knowledge of the blood types was available.

There were 50 ABO compatible and 18 incompatible grafts. Included with the compatible group were five cases of unknown blood types who received corneas from 0 donors (universal donor). When Rh typing was taken into consideration, 23 grafts were classified as incompatible and 40 as compatible.

RESULTS

Seven ABO incompatible and 15 ABO compatible grafts were successful. There were nine ABO or Rh incompatible and 12 ABO and Rh compatible successes. (See tables 1, 2, 3 and 4.)

Success was defined as the clarity of the graft and/or a postoperative visual acuity of 20/70 or better with a minimum follow-up of four months. Those classified as successful but with a visual acuity of less than 20/70 had an optically clear graft, but a definite other cause for the diminution of the visual acuity such as cataract or optic atrophy. The maximum follow-up of the

TABLE 3

SUCCESSFUL CASES

	Age (yr.)	Lamellar (L) or Penetrating (P)	Diagnosis	Recipient Type	Donor Type	Preop. Vision	Result	Follow-up (mo.)
ABO incompatible successes								
EB	45	P	Herpes	A+	B+	20/200	clear & 20/20	7
OB	40	P	Trachoma	O+	B+	20/200	clear & 10/200	16½
RE	53	L	Groenow's	O+	A+	CF	clear & 20/20	15¼
ET	18	P	Keratoconus	A+	AB−	20/200	clear & 20/25	17
JT	36	P	Leukoma	O+	B−	HM	clear & 20/60	7½
CW	62	P	Trachoma	O−	B+	CF	clear & 3/200 (cataract)	5½
VJ	36	P	Interstitial Keratitis	A+	AB+	20/80	clear & 20/70	4
ABO compatible successes								
WB	48	P	Hypopyon Keratitis	O+	O+	20/100	clear & 20/70	10
EB	74	P	Opacity	A−	A+	HM	clear & 20/100	6½
NB	34	L	Herpes zoster	A+	O−	HM	clear & 20/30	8
GD	64	L	Pterygium	O+	O+	CF	clear & 20/60	12
MH	47	P	Keratoconus		O−	20/200	clear & 20/60	6
VJ	55	P	Interstitial Keratitis	A+	O+	12/200	clear & 20/70	8½
LM	43	P	Traumatic ulcer	O+	O+	HM	clear & LP (phthisis)	4¾
AP	70	L	Leukoma	O+	A+	HM	clear & CF (optic atrophy)	4
RR	55	L	Previous transplant, ulcer	O+	O+	HM	clear & 20/200 (cataract)	14¼
OR	78	L	Herpes	B+	B+	HM	clear & 20/70 (cataract)	8
ER	48	P	Interstitial Keratitis	A+	A+	CF	clear & CF (cataract)	6
RR	37	P	Leukoma	O+	O+	HM	clear & 20/70	16
ES	57	P	Fuchs'	O+	O+	<20/200	clear & 20/70	17
HW	10	P	Herpes	B+	O+	LP	clear & 20/200	5
EG	30	L	Mooren's	O+	O+	HM	clear & 20/70	18

TABLE 4

FAILURES

	Age (yr.)	Lamellar (L) or Penetrating (P)	Diagnosis	Recipient Type	Donor Type	Preop. Vision	Result	Follow-up (mo.)
ABO incompatible failures								
LC	72	P	Fuchs'	O+	A+	HM	Opaque & HM	9½
EG	30	P	Ulcer	O+	A−	CF	Opaque & HM & repeat transplant	1
EG		P	Previous transplant	O+	B+	HM	Enucleated	
EH	69	P	Fuchs'	A+	B+	HM	Bulging & repeat transplant	3
AH	69	P	Keratitis	B+	A+	CF	Opaque & HM	1½
ML	69	L	Mooren's	B+	A+	LP	Ectatic, LP, enucleated	¾
GM	72	P	Fuchs'	A+	AB+	HM	Opaque & LP	¾
ER	54	L	Herpes	O+	B+	CF	Opaque & LP	16
KW	61	P	Trachoma	A+	B+	LP	Opaque, LP, & repeat transplant	9
JW	53	L	Previous transplant, trachoma	B+	A+	HM	Ectatic, vascularized & LP	7
MW	72	P	Trachoma	O+	B+	LP	Opaque & LP	1
ABO compatible failures								
CA	32	P	Keratoconus	A+	A+	CF	Opaque & LP	2
WB	63	P	Previous transplant, trachoma	O+	O+	HM	Opaque & necrotic	¾
WB		L	Previous transplant, trachoma	O+	O+	<HM	Opaque & LP	6¼
OB	28	P	Keratoconus	O+	O+	HM	Opaque & HM	1¾
OB	40	P	Trachoma	O+	O+	2/100	Opaque & 20/200, repeat transplant	4
AC	39	L	Previous transplant, leukoma	B+	B+	CF	Opaque & CF	14
RD	62	P	Descemetocele	A+	A+	HM	Opaque & HM	1
FE	57	L	Herpes zoster	A+	O+	LP	Opaque & LP	3¼
ME	56	L	Herpes	O+	O+	CF	Opaque	¾
JF	69	P	Hypopyon ulcer	A+	A−	HM	Opaque & CF	1
VF	56	L	Abscess		O+	CF	Opaque & CF	8
GH	36	L	Keratitis	A+	A+	HM	Opaque & repeat transplant	¼
GH		P	Previous transplant, keratitis	A+	A+	<HM	Opaque & CF	1
EH	66	P	Fuchs'	A+	A+	CF	Opaque & HM	14
EH		P	Previous transplant, Fuchs'	A+	O+	HM	Opaque & CF	27½
EH		P	Previous transplant, Fuchs'	A+	O+	LP	Opaque & LP	2¼
WK	67	P	Opacity due to endophthalmitis	A+	A+ (self)	HM	Opaque & HM	6
WK		P	Previous transplant	A+	A+	HM	Opaque & HM	16
ML	69	P	Mooren's	B+	B+	HM	Opaque & LP	6
DM	17	P	Congenital dystrophy	A+	A+	CF	Opaque & CF	12
JM	2	P	Descemetocele		O+		Opaque	6
WM	68	P	Previous transplant, Fuchs'	O+	O+	LP	Opaque & LP	10
EN	71	P	Previous transplant, keratoconus	A+	O+		Opaque & HM, enucleated	2
HP	63	P	Trophic ulcer	A+	O−	LP	Opaque & LP	15¼
FS	64	P	Chemical burn	A+	A+	LP	NLP, slough, enucleated	¼
AS	50	P	Previous transplant, keratitis	A−	O−	CF	Opaque & HM	13
SS	69	P	Trachoma	A−	O−	20/200	Opaque & CF	27
SS	68	L	Descemetocele	B+	O+	20/400	Opaque & HM	23
GS	69		Mooren's	A+	O+	HM	Opaque	12
AT	43	P	Leukoma		O+	HM	Opaque & CF	8½
AV	73	P	Ulcer	A+	O+	HM	Opaque & HM	2
JW	57	L	Herpes	AB+	AB+	HM	Opaque & HM	2
WW	63	P	Herpes	A+	A+	HM	Opaque	7
AW	52	P	Opacity		O−	LP	Opaque & HM	14½
MW	48	P	Keratoconus	O+	O+	CF	Opaque & HM	2

successful transplants was 1.5 years.

In the ABO compatible group 15 (30 percent) transplants were successful as compared to seven (39 percent) of those incompatible and 32 percent of the entire series. Similarly on the basis of ABO and Rh factors, 12 (30 percent) of the compatible series were classified as successful as were nine (39 percent) of the incompatible group. Of the 22 successful transplants in the total series, seven were ABO incompatible, nine ABO or Rh incompatible (tables 1, 2, 3, 4). Failures were those cases which did not meet the above criteria of clarity of the graft or visual acuity. Opacification or sloughing of the graft or retransplantation or enucleation of the eye at any point during the follow-up was considered failure. The failures included those grafts which were of benefit to the patient in retaining his eye or relieving his symptoms, but became opaque. Failures occurred in essentially the same incidence in the compatible (70 percent), and incompatible (61 percent) groups as in the entire series (68 percent).

Discussion

The 32 percent over-all success rate in this study is comparable to other larger series. The pooled results of several centers for 362 corneal grafts with four months or more follow-up revealed a 36.5 percent incidence of clarity and 36.2 percent of improvement in visual acuity to 20/200 or better.[13] Stansbury[14] reported that only 13 percent of 165 transplants, followed for a period of six months or more, had a postoperative visual acuity of 20/200 or better. Paton, [15] however, with a minimum follow-up of two months, obtained 64.5 percent clear grafts in 299 cases and 61.6 percent postoperative visual acuity of 20/200 or better in 185 cases.

In the present series it is of interest that all transplants resulting in 20/25 or better vision were in the ABO incompatible group and all those with 20/40 or better were in the ABO or Rh incompatible group. In the former group, there were three cases with 14 months or more follow-up, two of which had 20/25 or better visual acuity.

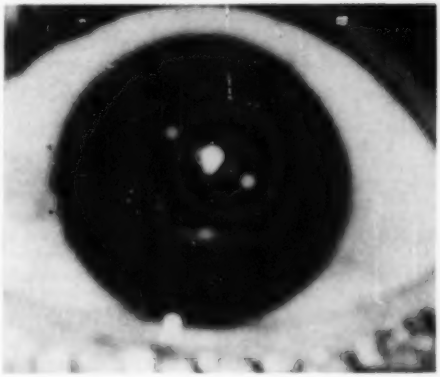


Fig. 1* (Mehri, Becker, and Oglesby). Patient OB (blood type 0+) with trachomatous scarring, O.S. Appearance of opaque graft from "compatible" (0+) donor two months after transplantation.

One particularly revealing patient is OB, type 0+, with trachoma diagnosed in 1930, and visual acuity of 2/100. A penetrating graft from an 0+ (compatible) donor was performed on the left eye on September 5, 1956: it became opaque with visual acuity at hand movements. A penetrating graft from a B+ (incompatible) donor was performed on January 30, 1957; it has remained clear for over 16 months. Visual acuity is 10/200 because of a cataract (figs. 1 and 2). The patient received topical steroids after each transplant.

Steroid medication may well be a significant factor in the success of transplants in incompatible cases. However, of the six successful transplants that received no steroids, three were classified as compatible and three as incompatible. It is of further interest that two of the incompatible successes that did not receive steroids had resultant visual acuity of 20/25 or better. The data on steroid treatment in the successful grafts are compiled in Table 5.

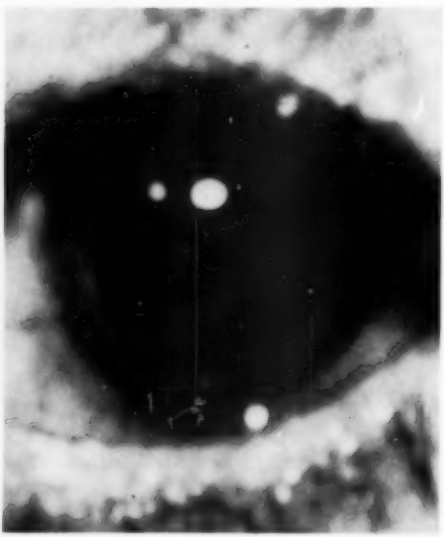
Havener, et al.,[16] in a series of 21 trans-

* Figures 1 and 2 were kindly supplied by Dr. Paul Miles.

TABLE 5

STEROID DATA

	Steroids Started (days postop.)	Steroids (dosage and duration)
		1. Incompatible Successes
EB		None
EB		None
NB	4	Topical Neo-hydeltrasol 5 times/day for 2 weeks; hydrocortisone 0.5% q.i.d. for 2 weeks; b.i.d. for 7 months
OB	115	Topical Neo-hydeltrasol q.d. for 1 year
RE	32	Topical Neo-delta-cortef b.i.d. for 3 months
VJ	4	Topical Neo-delta-cortef b.i.d. for 3 months; 0.5% hydrocortisone t.i.d. for 1 month; Neo-hydeltrasol continuing
ET		None
JT	14	Topical 0.5% cortisone q.i.d. for 2 weeks; discontinued for 2 months; then cortisone 0.5% t.i.d. for 2 months
CW	2	Topical Neo-hydeltrasol q.i.d., ? for how long
		2. Compatible Successes
WB		None
GD	9	Topical Neo-cortef 6 times/day for 2 days; Neo-hydeltrasol q. 2 h. for 8 days
MH	35	Systemic prednisolone 5 mgm t.i.d. for few days; topical Neo-deltrasol t.i.d. for 2 months and then at intervals
VJ	12	Topical Metimyd q.i.d. for 3 months; hydrocortisone 0.5% t.i.d. for ? 5 months; Neo-delta-cortef for 1 month; hydrocortisone for 1 month; Neo-delta-cortef continuing
LM	11	Systemic Meticorten 5 mgm b.i.d. for 3 days; systemic ACTH gel 100 units intramuscularly for 2 days; topical cortisone 0.5% q.i.d. for 1.5 months
AP	2	Topical Neo-cortef for 1 month
RR	7	Topical Neo-delta-cortef q.d. for ? 6 weeks
OR	8	Topical Neo-cortef 1.5% 6 times/day for 11 days; Neo-hydeltrasol q. 2 h. for 9 days
ER	7	Topical 2.5% cortisone q.i.d. for ? how long
RR	14	Topical Neo-cortef 5 times/day for 3 weeks
ES		None
HW	60	Topical 0.5% Metimyd q.i.d. for ? 2 months
EG		None



plants, found that three of 17 ABO compatible grafts and all four incompatible grafts became cloudy. They advocate the use of blood type compatible donor material only for corneal transplants. Nelken, et al.,[17] also suggest that only blood group-compatible corneas be used. Franceschetti[18] and Thomas[91] attach no significance to the donor-recipient blood type in corneal grafting.

In the present series ABO or Rh incompatibility did not seem to be a deterrent to the success in transplantation. The following possible explanations may be offered:

1. Late clouding of the homograft is unrelated to immunity.

Fig. 2 (Mehri, Becker, and Oglesby). Same patient as in Fig. 1 but two months after retransplantation, O.S., demonstrating clear graft from donor of incompatible blood group (B+).

2. Late clouding is related to immunity, but
   a. Antigens other than ABO or Rh antigens are responsible.
   b. Other conditions, such as a highly vascularized bed, [1,20] are necessary in addition to ABO or Rh incompatibility.
   c. The ABO-Rh titer of the incompatible successes may have been below a critical level.

Summary

Donor-recipient blood type incompatibility did not alter the incidence of success in a series of 68 corneal transplants. There were 22 successes (32 percent) in the entire series as compared to 39 percent of the incompatible group and 30 percent of the compatible cases.

640 South Kingshighway (10).

Acknowledgements

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BETA RADIATION*

As an adjunct to glaucoma surgery in the Negro

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The lamentable results obtained from surgery on Negroes with glaucoma are well known. According to Iliff,[1] sclerectomy is totally unsuccessful, trephination is about 23 percent successful, and iridencleisis about 50 percent successful. Despite this figure of about 50 percent success for iridencleisis, a number of surgeons feel that that operation for glaucoma on the Negro accomplishes little and they operate reluctantly and only when they believe that there is no alternative.

Glaucoma surgery in the Negro presents several special problems:

1. Procrastination

The average Negro patient with glaucoma applies for treatment much later than the average white glaucoma patient. Since most of these cases are of chronic simple glaucoma and without pain, the great majority of patients get several pairs of glasses in their search for visual aid and, in the meantime, permit the glaucoma to become advanced befor applying for medical aid. When these patients with well-advanced cases do apply for aid, it is often too late for either medicine or surgery.

2. Keloid formation

This tendency is frequently considered the cause of surgical failure. It should be borne in mind that keloid formation, a condition in which there is a major overgrowth of scar tissue initiated by trauma, is most commonly seen in the second decade and diminishes thereafter, the process ordinarily disappearing by the age of 50 years. Since most of our patients are over 40 years of age, keloid formation is hardly an important factor.

3. Color of the iris

Venable[2] has pointed out that pigmentation of the iris is definitely a factor in precipitating glaucoma. He found that both narrow- and wide-angle glaucoma are more fulminating in the dark-skinned Negro with deeply pigmented iris. The mulatto responds favorably to miotics and his glaucomatous course is favorable. The dark, heavily pigmented iris is harder to dilate and also to constrict. One patient of Venable's, a dark-skinned Negro with blue irises, responded favorably to surgery and required no postoperative miotics. Venable states that he has never seen glaucoma develop in a Negro albino. These findings suggest very strongly that the basic causative factor of failure of surgery in the Negro may be the increased amount of pigment present in the iris.

4. Provocative tests

Insofar as pressor congestion tests and rise of tension following dilation are concerned, the results in the Negro race are essentially the same as those in other races.

Technique of operation

The operation to be described is one that evolved. It began as a modified Lagrange operation and has been modified in the following manner:

When iridencleisis became an established procedure, it was included; after Masters and others established the ab externo incision technique, that also was included. Following a conversation with Dr. Kronfeld, the use of scissors to remove part of the anterior lip of the incision was replaced by use of a Holth punch. Dr. Kronfeld stated that the pathologic examination of a number of eyes in which an Elliot trephination had failed, showed a common fault. In all of

* From the Wills Eye Hospital.

these failures, the small trephine opening showed ridges, as if the operator had stopped several times to look at the cut instead of making a continuous cut with firm pressure. He felt that these ridges could have been initiating factors in producing failure of drainage, as they might have started a fibrotic process which occluded the opening. A punch produces a clean-faced, sharp-edged wound and is very easy to use.

The operative procedure used is:

The conjunctiva is grasped by a forceps near the insertion of the superior rectus and a limbus-based flap containing conjunctiva and Tenon's capsule is dissected in one unit, obtaining as clean a sclera as possible. The conjunctiva is then split over the cornea for a distance of 1.5 to 2.0 mm. from the limbus.

Using tension sutures on both sides of the wound, a keratome is used to scratch an incision about 6.0- or 8.0-mm. long in the sclera 1.5 mm. from the limbus. Usually, after this, by traction and tension with the anterior suture, the iris can be prolapsed. If the iris does not prolapse, an iris forceps is inserted and the iris is brought out. The iris is then grasped by the operator with another pair of forceps, and an assistant cuts the iris between the two pairs of forceps. The iridencleisis then is performed.

Using a scleral punch, one or two snips are made in the anterior lip of the wound. After this, the conjunctiva is sutured by interrupted sutures and a drop of one-per-cent atropine solution is instilled in the eye, the eye is bandaged, and the patient returned to his room. Follow-up consists of daily massage and sometimes atropine at the first dressing. The patient is usually discharged in five to seven days after the operation.

This operation gave a very high percentage of good results in white patients and we have used it in its varying forms over a number of years. However, when performed upon Negro patients, the results were just as unsatisfactory as any other standard procedure. It is difficult to obtain figures on failures in the Negro for several reasons: (1) Statistically, reports are usually not according to race and (2) surgeons are reluctant to report failure, which is what so often follows glaucoma surgery upon the Negro.

Because of the high failure rate, it was felt that perhaps some improvement or addition to the previously described operation might be of value. Among the possibilities was beta radiation. Although beta radiation has been used for ocular therapy, its use is usually limited to superficial lesions. Haik[3] and others used beta radiation in glaucoma with the idea of reducing the vascularity of the ciliary body and thus its secretion. They were unsuccessful.

Strazzi[4] used radiation therapy for relief of pain in absolute glaucoma. Toniolo[5] also used radiation. Haik[6] and others used it on a one-eyed five-year-old boy months after the operation, with some improvement. In the series to be described, beta irradiation was used as a planned part of the operative procedure, on useful eyes in a continuous series of cases, the main purpose being to preserve vision rather than to relieve pain in blind eyes.

The thought that prompted the use of beta irradiation was to increase the effectiveness of a proved filtering operation. With this in mind, beta irradiation was applied to the operative site immediately following surgery.

The manner in which beta irradiation acts is not known but from the apparent results of this short series, it seems to have aided in retarding some factor that hindered filtration.

The appearance of the end-result is identical to that following the same surgical procedure without radiation. The appearance of the bleb is not altered and the adjacent tissues seem unaffected, both soon and a longer time after the application.

Beta radiation

The biologic effects of beta irradiation,

insofar as we know, are the same as those of radium and X-ray therapy. They differ only in the depth of the tissue irradiated. It should be recalled that radiation is always destructive. The difference in sensitivity of cells is the basis of all radiation therapy. Established X-ray and radium therapeutic procedures should apply to beta irradiation, provided that the effect desired is superficial. The present approach is, we feel, based on established principles of radiation therapy.

Radiation therapy to prevent or retard the overgrowth of normal tissue has been used by radiologists for many years. A well-known example of this is its use in the treatment of keloid growths. A well-developed thick keloid will not respond to X-ray therapy in safe dosage. However, if the keloid is first removed surgically and then the site of the excision is treated either with radium or Xrays, the scar often remains flat. This effect is due to the greater radiosensitivity of the young fibroblasts. This depression of fibroblastic activity is the basis for treatment in the present cases. In this series an attempt has been made to prevent the obliteration of the operative fistulas with the use of beta irradiation (Sr^{90}). The depth dose measurements for the strontium^{90} applicator* used in these cases were: 50 percent at one mm., 25 percent at two mm. and nine percent at three mm.

In planning therapy with beta irradiation our first problem was how much beta to use, and the second, the optimum time of treatment. Because of previous experiences with the treatment of early newly formed blood vessels in the cornea and the relatively equal ratio of sensitivity of new vessels and young fibroblasts, it was decided to use this same dosage schedule. Our treatment for corneal vascularization has always been fractionated —2,100 rep are given per treatment, repeated usually at weekly intervals. Occasionally the treatments are given twice a week. Three or four treatments are usually sufficient. A total dosage is 6,000 to 8,000 rep.

According to Hughes,[7] "applying more than 3,000 rep tends to produce lens opacities." In our series to date, we have not seen any lens opacities develop, any damage to the epithelium, or any unpleasant subjective symptoms occur that were due to radiation.

As to the optimum time of treatment it was decided that the beta irradiation should be given at the time of operation so that its effect would be utilized as soon as repair began. No advantage could be seen in giving preoperative irradiation since fibroblastic activity had not yet begun.

The treatment was given in the operating room; 2,100 rep were applied to the surgical incision at the finish of the operation. This treatment was repeated at weekly intervals for a total of three treatments—6,300 rep. In two of the cases only one treatment was given. This dosage schedule is by no means final. Subsequent cases may show that shorter intervals between treatments may produce better results or that a single treatment may be sufficient.

Case reports

Cases 1 and 2

O. S., a Negro, aged 40 years, was first seen on November 6, 1953. At this time, he complained of seeing "ribbons around lights and at times, flashes." When first seen, his visual acuity was: O.D., 6/9−1, corrected to 6/9+2; O.S., 6/9 corrected to 6/9. The tension in each eye was 30 mm. Hg (Schiøtz).

External examination of the eyes was essentially negative except that the right eye had a definite pericorneal injection and also a small scar of the cornea near the limbus at the 4-o'clock position. The left eye exhibited no external pathologic alteration. His general physical examination was negative except for mild hypertension at times (160/95 mm. Hg).

Because of contracted pupils, his fundi could not be seen well but no gross pathologic process was noted. The slitlamp revealed some endothelial deposits but otherwise was negative. Gonioscopic examination showed narrowed angles in both eyes

* Technical factors of the Sr^{90} applicator used in this series:
Active diameter—7.8 mm.
Filtration—2.0 mils of stainless steel; 10 mils of aluminum.
Output—36 roentgen-equivalent betas per sec.
All treatment is contact therapy.

and the rate of outflow in each eye was 0.08. The fields of both eyes were quite contracted.

He was placed on miotics, beginning with 0.5-percent pilocarpine nitrate and increased to 2.0 percent pilocarpine nitrate, 1.0-percent eserine, and also Diamox. Since this failed to control his tension and his fields were diminishing, surgery was advised and he consented to have his left eye operated upon. The operation described was performed with apparent success.

One month later, the tension in the right eye became rapidly elevated to 45 mm. Hg despite miotics and Diamox, and he agreed to have this eye operated upon. This was done and the pressure has remained in the low twenties, or lower, since that time without medication. The observation period has been over a year and there has been very little field loss. The visual acuity is: O.D., 6/15 corrected to 6/15; O.S., 6/9 corrected to 6/6.

Case 3

J. P., a Negro, aged 62 years, was first seen on April 19, 1955. At this time he stated that his left eye had been pink for over five weeks and was steadily getting worse. It was moderately painful and things seemed smoky. His visual acuity was: O.D., 6/60 corrected to 6/9+2; O.S., 5/60 corrected to 6/60.

Upon examination, the right eye was essentially negative except that the pupil was slightly dilated and reacted poorly to light and convergence. Finger tension in this eye was normal. In the left eye, there was a definite pericorneal injection, the pupil was larger than that of the right eye, the pupil was elliptical in shape and showed almost no reaction to light and convergence. The finger tension of the left eye was elevated and tonometric readings were: O.D., 27 mm. Hg (Schiøtz); O.S., 49 mm. Hg. His general history and physical findings were negative except for some deformity and interference with motion of the joints of the hands, elbows, and knees.

Because of the corneal haze, no details could be made out in the fundus of his left eye. Since the anterior chamber seemed shallow, the diagnosis was acute exacerbation of chronic narrow-angle glaucoma and he was admitted as an emergency case. He was given a course of mecholyl and prostigmine, Diamox, and 2.0-percent pilocarpine nitrate and eserine. Despite this treatment, the tension remained at 45 mm. Hg.

Slitlamp and gonioscopy revealed nothing additional except some old crenated keratic precipitates in the left eye and narrow angles in both eyes. There was no flare in the left eye and the iris appeared normal. Tonography of the right eye when the tension was 15 mm. Hg gave a coefficient of outflow of 0.26; on the left eye with the tension at 40 mm. Hg, coefficient of outflow of 0.13. With a 20/1,000 examination, the field of the left eye was approximately round and 10 degrees in size and was reduced to about five or six degrees when examined with a 5/1,000 test object.

Surgery was advised and performed on April 26, 1955. Since then, the patient has been followed carefully. The pressure has remained soft—about 10 to 11 mm. Hg—and there has been no noticeable field loss.

Case 4

S. Y., a Negro, aged 42 years, was first seen in 1945. At that time his diagnosis was traumatic cataract in the left eye. The visual acuity was: O.D., 6/30—1; O.S., hand movements.

When seen on August 18, 1950, his only complaint was of a gritty feeling in his right eye. The external examination was essentially negative and he responded to a mild eye wash. In March, 1951, a cataract extraction was performed on the left eye and the tension in the right eye was 30 mm. Hg. Despite miotics (two-percent pilocarpine) at this time, the tension in the right eye was erratic, going as high as 49 mm. Hg. This patient co-operated poorly on a medical regime, not reporting regularly as requested. The pressure usually varied from 20 to 40 mm. Hg. In March, 1954, when the pressure was 40 mm. Hg on miotics, surgery was strongly advised and accepted.

The external examination of the right eye was negative with the pupil contracted. Details of the fundus could not be made out because of a hazy lens. The slitlamp revealed some iris atrophy and ectropion uveae in the right eye. With gonioscopy, the angle was found to be open and the trabeculas could be clearly seen.

The 20/1,000 field of the right eye was about four degrees concentrically contracted around the macula. The coefficient of outflow was: O.D., 0.11; O.S., 0.05.

On March 22, 1955, the iridencleisis already described was performed, with no complications other than a small persistent hemorrhage in the right eye. He was discharged with a soft tactile tension and without any medication in this eye. Within two months, however, even with miotics, the pressure fluctuated between 22 and 40 mm. Hg but with no field loss. Inasmuch as there was no real control of tension, this case is considered a failure.

Case 5

F. C., a Negro, aged 47 years, was first seen in January, 1955, complaining of a swollen right upper lid. At this time, vision was: O.D., 3/60, corrected to 3/60; O.S., 6/60, correctible to 6/9+2.

The cause of the swelling was a chalazion of the right upper lid but a mild conjunctival injection was also present in the right eye. The pupil of the right eye was slightly larger than the left (3.0 mm. vs. 2.0 mm.) but both reacted to light. The tension in each eye was 40 mm. Hg. This patient had had no previous treatment but had noticed dimness of vision in the right eye for some time.

The fundus of the right eye could be seen only poorly because of hazy media but there was definite glaucomatous cupping. Details of the left eye could not be made out because of the small pupil. Slitlamp examination of the right eye showed some deposits on the anterior surface of the right lens

but no pathologic process in the left lens and, since both angles were narrow, the diagnosis was narrow-angle glaucoma of both eyes, with evidence of inflammation in the right eye. The field in the right eye was contracted with macular involvement; in the left eye the field with 3/330 was approximately 25 degrees in size. In the right eye the coefficient of outflow was 0.03 with an initial pressure of 59 mm. Hg; in the left eye, 0.10 with an initial pressure of 37 mm. Hg.

He was admitted for treatment but could not be controlled medically; therefore, surgery was advised and performed. The operation was uneventful but, following surgery, there was some hemorrhage into the anterior chamber. This absorbed without difficulty and the eye remained quiet with the pressure controlled for a time.

When seen on March 29, 1955, however, the pressure had risen to 53 mm. Hg. Following this, the patient transferred to another hospital.

CASES 6 AND 7

F. R., a Negro, aged 62 years, was first seen on April 9, 1954. At that time he stated that his left eye had been sore and inflamed for the past four or five days. When examined, the left eye was found to have a mild pericorneal flush with a tension of 49 mm. Hg and bedewing of the cornea. The right eye appeared normal. As far as could be ascertained, this was his first attack of glaucoma and his past medical history was negative for eye trouble.

His general physical examination was negative except for hypertension of 180/110 mm. Hg. With the slitlamp, bedewing of the cornea was seen but no cells were seen in the anterior chamber and the iris was quiet. With the gonioscope, the right eye showed a narrow but open angle with no synechias. The ciliary zone not visible (the trabecular spaces open). In the left eye, the trabecular spaces were very shallow but no synechias were present. The angle was narrow but there were engorged vessels in the angle which, at times, formed small convoluted raised balls.

Because of these findings the diagnosis was acute narrow-angle glaucoma in the left eye. Both fields were full with a 3/330 test object.

The patient was admitted and put on a medical regime. The frequent use of miotics every three hours around the clock and Diamox were required to keep the tension under 30 mm. Hg. Because of this, surgery was felt necessary, advised, and performed on the left eye in April, 1954. Recovery was uneventful except for the presence of a few flecks of hemorrhage which absorbed. However, during convalescence, despite the instillation of pilocarpine in the right eye, there was a sudden elevation of tension in this eye and the patient elected to be operated upon. This was done and nothing untoward occurred. He was followed until November, 1955, at which time both fields were full. Vision was: O.D., corrected to 6/6; O.S., corrected to 6/9—2. Miotics were not required. The tension in both eyes was 15 mm. Hg.

CASE 8

E. R., a Negro, aged 56 years, was first seen in April, 1950. At the time, he complained of failing vision in his left eye. Visual acuity was: O.D., 6/21 correctible to 6/6; O.S., 6/60 correctible to 6/15. The external examination was negative, with both pupils contracted. Tension was: O.S., 23 mm. Hg; O.D., 36 mm. Hg.

Nothing pertinent was found in a general physical examination and history. The slitlamp examination was negative except for some pigment particles on the lens. With the goniolens, numerous anterior synechias and marked pigmentary deposits were seen throughout the angles, which were narrow. At this time, the field in the left eye showed loss of the nasal field and encroachment of the macula.

Under medical treatment, the tension was kept in the 20 to 30-mm. Hg range until early in 1954 when it could no longer be controlled by medication.

On March 12, 1954, he was operated upon and on March 20, 1954, he was discharged from the hospital, both operation and recovery having been uneventful. However, when re-examined on March 31, 1954, there was evidence of uveitis and he was re-admitted to the hospital. Under treatment this cleared up and, since then, the tension has remained under 20 mm. Hg without miotics and no appreciable field loss has been noted. There was recurrence of uveitis in the left eye with great loss of field and vision.

CASE 9

W. B., a Negro, aged 52 years, was first seen on September 30, 1955. At that time, his visual acuity was light perception in the right eye and 6/12 correctible to 6/9 in the left eye. At the time, the patient stated that he had lost the vision in his right eye during the past year without any pain. He now noticed a dimness of vision in his left eye. This was his first attempt to do anything about his eyes.

Other than for the presence of arcus senilis, the external examination was negative despite both eyes being hard, with a tension of 55 mm. Hg in each. He was advised admission to the hospital but refused, whereupon he was put on a medical regime of 2.0-percent pilocarpine and 0.5-percent eserine in both eyes, three times daily. This did not control the tension and he was admitted to the hospital in October. Insofar as his eyes were concerned, his past medical history and physical condition seemed negative.

The fundi of both eyes showed marked glaucomatous cupping and pallor and a blood vessel ratio of 1:2 with some arteriovenous compression. The maculas of both eyes seemed granular. The slitlamp examination was negative except for some nuclear sclerosis in both lenses. With the gonioscope, both eyes seemed to have open angles but, in the left eye, there was an anterior synechia extending to Schwalbe's line. The tonometer reading was 0.11.

The field of the left eye was about five degrees around with a 1/1,000 examination. In view of this very small field, we were reluctant to operate but,

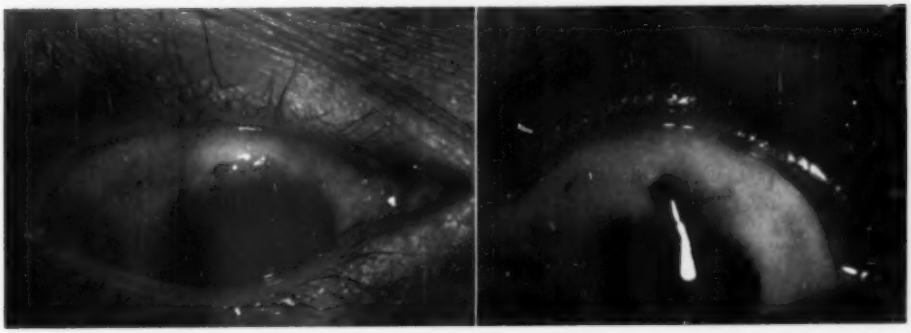

Fig. 2 (Cohen, et al.). Cases 9 and 10 are illustrated to show that no keratinization of the conjunctivas occurred.

after due deliberation and consultation, surgery seemed justified since it was felt that the eye would be lost otherwise, as was the right eye. The operation was performed and the pressure has remained low—about 15 mm. Hg—without miotics.

CASE 10

D. G., a Negro, aged 48 years, was first seen on October 11, 1955. At that time the patient stated that vision in his left eye had been poor for several months. Vision was found to be 6/6 in the right eye and 6/60 in the left eye. The external examination was essentially negative except that the left pupil was markedly larger than the right and did not react to light and accommodation. The finger tension was elevated, 48 mm. Hg in the left eye as compared to 26 mm. Hg in the right eye.

Ophthalmoscopic examination of the right eye revealed a normal fundus, the disc being round, having a good color, and only a small cup. In the left eye, the disc was round with deep glaucomatous cupping, numerous pigmentary deposits adjacent to the temporal portion of the disc, and some pigmentary disturbances in the macular region. The peripheral portion of the left eye was negative.

The patient gave a history of trauma to the left eye at the age of nine years, followed by a permanently reduced vision in this eye. When examined by slitlamp, neither eye showed any flare or keratic precipitates. However, the left eye showed a star-shaped central opacity which was considered to be a possible Vogt-type contusion cataract. With the goniolens, the right eye had an open angle and no pathologic change. In the left eye, however, although the angle was apparently open, there was marked pigmentation, especially at the 6-o'clock position. Also present was a grayish membrane which was partially covered by pigment in the trabecular area. The grayish membrane seemed to cover and plug up the trabeculas and also, in places, covered the ciliary body. Only a very small amount of inferior nasal field remained with a 3/1,000 test object. The coefficient of outflow was 0.20 in the right eye and 0.03 in the left.

The patient was advised admission but delayed for four weeks, during which time the pressure in the left eye went to 62 mm. Hg. He was then admitted and treated medically, tension at first responding and staying in a range of 12 to 26 mm. Hg. After a few days the tension became elevated and persisted in the high thirties and operation was advised. This was performed and both it and recovery were uneventful. The visual acuity in the left eye following surgery was 3/60 and tension has remained under 20 mm. Hg without miotics. In 1957, the finger tension in the left eye was still normal.

COMPLICATIONS

1. *Presentation of vitreous.* In two of the cases, vitreous presented after the anterior chamber had been opened. The vitreous was cut off and the operation continued. The presentation of vitreous did not seem to affect the result.

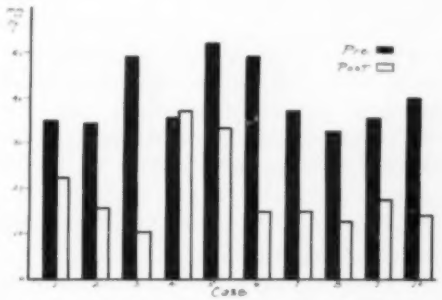

Fig. 1 (Cohen, et al.). Pre- and postoperative tensions.

TABLE 1

SURVEY OF CASES REPORTED

	Vision		Tension		Tonography	Fields		Observation Period	Gonioscopic Type ∠	Result
	Pre	Post	Pre	Post		Pre	Post			
1. O.S. O.S.	6/9 c=6/9	6/15	30–40	18–26	0.08	Small island near fixation	Retained c loss	14 mo.	Narrow	Improved
2. O.S. O.D.	6/9−1 c=6/9+2	6/6	23–45	12–21	0.08	Contracted Field	Retained c loss	13 mo.	Narrow	Improved
3. J.P. O.S.	6/50 c=6/60	6/9	49	10–11	0.13	Small field of about 5°	Retained	8 mo.	Narrow	Improved
4. S.Y. O.D.	6/30	6/60	30–40	22–48 c Mi.	0.11				Open	Unimproved
5. F.C. O.D.	3/60 c=6/6	Hem. in A.C.	40–59	14–48 c Mi.	0.03	Small central Macula involved			Narrow	Unimproved
6. F.R. O.S.	6/9 c=6/6	6/12 c=6/6	49	15		Full	Slight nasal loss	18 mo.	Narrow	Improved
7. F.R. O.D.	6/9 c=6/6	c=6/6	30–45	15		Full	Slight nasal loss	18 mo.	Narrow	Improved
8. E.R. O.S.	H.M.	C.F.	29–34	13		Indefinite	No change	14 mo.	Narrow	Improved
9. W.B. O.S.	6/12 c=6/9	6/30	36	18	0.11	Almost to fixation	No change	2 mo.	Open	Improved
10. D.G. O.S.	6/60	3/60	40	12–15	0.03	Small island near fixation	No change	2 mo.	Open c membrane	Improved

2. *Hemorrhage postoperatively.* At the first dressing, the anterior chamber is sometimes found to be full of blood. So far, the blood has absorbed completely without causing any difficulty or complications.

3. *Abnormal conjunctiva.* In two cases, on reflecting the conjunctiva, it was noted that it peeled unusually easily and felt stiff and somewhat parchmentlike. In one of these cases, the result was a complete failure and in the other a poor result was also obtained, suggesting that the permeability of the conjunctiva may also be a factor in determining the success of an operative procedeure.

4. *Development of cataracts postoperatively.* In no case, so far, using the surgical technique previously described, have we seen a cataract develop postoperatively in either white or Negro cases.

SUMMARY AND CONCLUSIONS

A small series of cases has been presented in which the described surgical procedure has given good results over an observation period of, in some cases, up to 18 months. Eight out of the 10 cases attempted have been successful, a higher ratio of success than is usually obtained.

Exactly what the addition of the beta irradiation does is not known. However, experimental work by other men, McDonald, et al.,[3] shows that it depresses fibroblastic activity in the cornea. Another possibility is a reaction of the chromatophores. Whatever the mechanism, it appears to be an effective addition when used after glaucoma surgery on the Negro. It is interesting that the results obtained were similar to those of Iliff[1] who used three fractionated doses instead of one at the time of surgery.

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STUDIES ON HERPES SIMPLEX VIRUS*

IX. Corneal responses to repeated inoculation with herpes simplex virus in rabbits

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Recurrent lesions of herpes simplex on the skin are a severe annoyance to the individual but are seldom incapacitating. Recurrent herpetic lesions of the cornea on the other hand frequently result in disastrous impairment of vision and are, at present, among the most important external eye diseases in the United States.[1] The basic immunologic characteristics of repeated herpetic lesions of the cornea are probably no different than those of recurrent herpes in other sites. Following the primary infection, systemic antibodies develop but in spite of them the local circumscribed lesions continue to be elicited by known or unknown trigger mechanisms. However, the avascular nature of the cornea makes this tissue somewhat independent of circulating antibodies, and the cornea itself can apparently form antibodies.[2] Thus corneal herpes presents some fundamental problems of interest as well as of obvious practical importance.

Unfortunately, there is as yet no experimental model of latent and recurrent herpetic eye infection available in the intact animal. Since Gruther's[3] demonstration in 1913 that the rabbit cornea provided a suitable site of inoculation for the virus of herpes simplex, several observers have studied the effect of repeated corneal inoculation of this virus. Thus Loewenstein[4] and Doerr and Vochting[5] were unable to induce corneal lesions by re-inoculating the virus two to six weeks after an initial take in the same eye but found no such resistance in the other eye of the animals. They concluded that local rather than systemic immunity accounted for the results. Hall, MacKneson, and Ormsby[6] found that re-inoculation of corneas one month after an initial infection of the eye with herpes simplex virus resulted in 42-percent incidence of herpetic keratitis which tended to be milder than the first attack. They also indicated that circulating antibodies did not protect against keratitis when virus was inoculated into a previously uninvolved eye.

All previous studies have employed a single re-inoculation of the cornea with the same virus as employed in the first infection. Differences in organotropism of strains of herpes simplex virus[7] and definite, although small, antigenic differences between strains have been recorded.[8,9] Therefore, it was of interest to study by quantitative methods the effects of repeated inoculation of the cornea of rabbits with different strains of this virus.

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Materials and Methods

Virus strains

The following strains of herpes simplex virus were employed:

a. HF mouse-adapted, originally obtained from the American type culture collection as the 38th mouse passage, used in the 65th mouse passage with an intracerebral LD50 for mice of $10^{-4.7}$.

b. H 51 mouse-adapted, received in the form of amnion cell tissue culture fluid through the courtesy of Dr. H. Ormsby and Dr. A. M. Fowle. It was carried by intracerebral mouse passage and had an LD50 of $10^{-4.0}$ by this route.

c. PET, a strain originally isolated from a primary infection in an adult[8] and subsequently carried as a mouse line an egg line. The mouse-adapted line was used in its 25th intracerebral passage and had an LD50 of $10^{-4.3}$ to $10^{-4.4}$. The egg-adapted line in the form of allantoic fluid was employed as the 60th yolk sac passage. It had an LD50 of $10^{-3.8}$ by yolk sac inoculation of 10-day embryonated eggs and an LD50 of $<10^{-1.5}$ for mice.

Experimental animals

Both white New Zealand and colored rabbits were obtained from a commercial source. For systemic immunization the animals were given four to six subcutaneous and one or more intraperitoneal or intravenous injections of mouse-adapted or egg-adapted PET virus in graded concentrations over periods of from three to six weeks.

Neutralization tests

At various times rabbits were bled by cardiac puncture, the serum separated and kept in the frozen state at $-20°$C. until the antibody content was assayed. Neutralization tests were performed by the constant serum-virus dilution method with intracerebral injection of the mixtures into 13 to 16 gm. white Swiss mice, as described in detail elsewhere.[8] The comparison of strain PET and H51 was carried out by a constant virus-serum dilution method.[8] The results were expressed as the amount of virus neutralized (log of LD50 virus titer with normal serum minus log of LD50 virus titer with test serum). The results of infectivity titrations and neutralization tests were reproducible to within 0.5 log.

Corneal inoculations

Rabbits were securely wrapped in a sheet of muslin and the cornea anesthetized with a few drops of 0.5-percent pontocaine. The cornea was scarified in a "cross-hatched" pattern by means of a sterile platinum scraper dipped into virus suspension or control fluid. The volume of virus-containing fluid thus inoculated was estimated at 0.01 ml. In order to obtain regularly "takes" it was necessary to employ virus in the form of 20-percent brain suspension or undiluted allantoic fluid. The infective titer of this virus suspension for mice or eggs was simultaneously determined. The development of corneal lesions and their regression was followed by daily gross inspection, examination by ophthalmoscope, and staining of epithelial lesions with fluorescein. The presence of conjunctival inflammation and exudate, and the extent and intensity of corneal involvement were recorded daily and graded comprehensively from negative ($-$) to ++++.

Experimental results

1. The effect of systemic immunization resulting in measurable antibody levels on subsequent susceptibility of the cornea.

Rabbits were injected repeatedly with viable PET virus of the mouse- or egg-adapted line. After obtaining serum for measurement of antibody level they were challenged on both eyes with one of the virus lines.

A representative experiment is summarized in Table 1. The serum antibody levels varied from 0.5 to 1.5 logs of virus neutralized but there was no evident relationship between the level of antibody measured and the reactivity of the cornea to subsequent

TABLE 1

SYSTEMIC IMMUNIZATION WITH STRAIN PET MOUSE OR EGG LINE AND CORNEAL CHALLENGE

Rabbit No.	Immunized With	Serum Antibody*	Challenged With	Corneal Reaction	
				Right	Left
1	PET mouse	0.5	PET mouse	0	++
2	PET egg	1.2	PET mouse	0	+
3	PET mouse	0.7	PET egg	++++	++++
4	PET mouse	1.1	PET mouse	++	+
5	PET mouse	1.5	PET egg	++	++
6	PET mouse	0.7	PET egg	+++	+++
7	PET egg	1.0	PET egg	++++	0
8	PET mouse	1.4	PET mouse	++	++

* Logs virus neutralized by serum taken one day prior to corneal challenge.

virus inoculation. With eight eyes challenged with each, there was a tendency for the egg-adapted line to result in a more severe corneal reaction (average grade 2.5) than the mouse-adapted line (average grade 1.2). As the egg-adapted line had an LD50 of less than $10^{-1.5}$ for mice by the intracerebral route compared to an LD50 of $10^{-4.3}$ for the mouse line it is clear that "virulence" for rabbit cornea and for mice appear to be unrelated. The experiment confirmed earlier work that systemic immunity failed to protect against herpetic keratitis in the rabbit.

2. The effect of corneal infection on subsequent susceptibility of the cornea to re-infection by the same or another strain of herpes simplex virus.

The data presented above suggested that circulating antibody of low or intermediate levels failed to influence the susceptibility of the cornea to subsequent infection with herpes simplex virus, at least as far as the PET strain was concerned. The next group of experiments attempted to evaluate the role of one corneal infection on the susceptibility of the cornea to a subsequent infection with the same or a different strain of herpes virus. A representative experiment is presented in Table 2. In a group of 15 rabbits inoculated with the mouse line of PET virus, 20 of 30 eyes developed typical corneal lesions and one animal died from encephalitis. The remaining animals were permitted to recover completely and were then challenged with the same pool of virus, of the same proven mouse infectivity.

Only seven of 28 inoculated eyes developed specific lesions, suggesting some form of "resistance" of the cornea. However, when these eyes were re-challenged with a different strain of herpes virus the sequence of events repeated itself:

Initially there was a high rate of "takes" and later re-challenge with the homologous strain resulted in fewer and milder lesions. The incidence of corneal reactions to the first and second inoculation with each strain is statistically highly significant, but the results of challenge with the heterologous strain are equally dramatic. There appears to be strain specific immunity. To what extent might it be associated with circulating antibody levels?

Serial serum specimens had been obtained from a number of the rabbits included in Table 2. In six of them sera were assayed for neutralizing antibody. Table 3 lists the titers in relation to the eye lesions observed in three of the challenge inoculations. Following inoculation with strain PET measurable antibody levels appear in five of six animals although one of them had no detectable eye lesion. This antibody did not seem to protect against challenge with strain H51.

The second inoculation of H51, however, results in far fewer and milder lesions in spite of little, if any, increase in antibody level. The inoculation with strain HF on June 10th resulted in only six takes and no

TABLE 2

A REPRESENTATIVE EXPERIMENT

	First Inoc. on Cornea	Second Inoc. on Cornea	Third Inoc. on Cornea	Fourth Inoc. on Cornea	Fifth Inoc. on Cornea
Date of Inoculation	Jan. 7	Feb. 13	Mar. 11	Apr. 8	June 10
Virus Strain Used	PET Mouse	PET Mouse	H51 Mouse	H51 Mouse	HF Mouse
LD_{50} of Virus (I.C. in mice)	$10^{-4.1}$	$10^{-4.4}$	10^{-4}	10^{-4}	$10^{-4.7}$
No. Eyes Inoc.	30	28*	28	28	28
No. Eyes with Take	20	7	22	10	6
Percent Take	66.6	25	78.6	35.7	21.4
Average Grade Value of Corneal Lesion	2.2+	1.4+	2.3+	1.2+	1.1+
X^2	8.4	14.0	8.8		
P	0.004	<0.001	0.003		

* Reduction in no. of eyes due to death of one rabbit from encephalitis.

consistent influence on the circulating antibody level. Thus antibody levels do not appear to account entirely for differences in the corneal reactions.

The marked contrast in results of challenge inoculations with strains PET and H51 suggest biologic and perhaps antigenic differences. In terms of intracerebral titration in adult mice the two strains appear virtually the same. Titration in tissue culture, employing two cell lines (human carcinoma HeLa, and mouse fibroblast L) yielded no consistent or significant differences. To ascertain possible antigenic differences between strains H51 and PET, detailed cross neutralization tests in mice were performed with strain specific antisera prepared in rabbits. The essential results are presented in Table 4. It is seen that at two levels of serum dilution the homologous virus is neutralized to

TABLE 3

TITERS IN RELATION TO EYE LESIONS

Date	Procedure	Rabbit No. 157	Rabbit No. 158	Rabbit No. 163	Rabbit No. 164	Rabbit No. 166	Rabbit No. 167
Jan. 4	Bleeding	0*	0	0	0	<0.4	<0.5
Jan. 7	Eye. inoc. with Strain PET	−† −‡	− ++	++ ++	+ ++	++ ++	+++ −
Mar. 8	Bleeding	0.8	<0.5	1.1	0	1.0	1.2
Mar. 11	Eye inoc. with strain H51	+ +	+++ −	++ +++	+++ +	− ++	+++ −
Apr. 8	Eye inoc. with strain H51	− −	− −	− −	− +	+ −	− +
May 8	Bleeding	0.8	1.3	1.1	2.2	1.4	2.1
June 10	Eye inoc. with strain HF	− −	− −	− −	− −	− −	− +
June 24	Bleeding	1.3	<0.5	2.7	2.1	>1.7	1.0

* Log virus neutralized by serum.
† Lesions on right eye graded − to ++++.
‡ Lesions on left eye graded − to ++++.

TABLE 4

AMOUNT OF VIRUS NEUTRALIZED BY HOMOLOGOUS AND HETEROLOGOUS ANTISERUM

Rabbit Antiserum to Strain	Serum Diluted 1:9 Logs Virus Neutralized*		Serum Diluted 1:27 Logs Virus Neutralized*	
	H51	PET	H51	PET
H51	1.1	0.3	1.1	0.5
PET	1.0	1.3	0	0.7

* Average figures from two experiments.

a greater extent by each antiserum than the heterologous virus. This tends to support the impression that slight but definite strain differences are demonstrable between the two viruses employed in this work.

It was of interest to determine whether nonspecific "resistance" of the cornea might be induced by repeated inflammation. A series of rabbits, similarly treated to those shown in Table 2, exhibited fewer corneal lesions after each re-inoculation with herpes virus. However, when challenged with vaccinia virus all inoculated eyes promptly developed marked keratitis. Thus the falling reaction rate to herpes appeared to be associated with some specific resistance to that agent.

DISCUSSION

In recurrent herpetic keratitis in man it is often, but not always, true that the inflammatory reaction is less and less marked with each successive attack. However, because each attack leaves residual scar tissue, there is increasing opacification of the cornea and progressive loss of vision. The presence of significant titers of circulating antibody obviously does not interfere with recurrent herpetic lesions in either skin or eye. Similarly the presence of circulating antibody to herpes simplex virus failed to have any influence in the experimental infection of rabbits, and the titer of specific antibody was unrelated to the severity of corneal lesions resulting from challenge infection.

On the other hand earlier investigators have pointed to a certain "resistance" of the rabbit cornea following initial infection with herpes simplex virus.[1, 5, 6] These earlier studies utilized only a single strain of herpes simplex virus for both the initial infection and the challenge of the eye. From these results it was concluded that the "resistance" to re-infection presented, in fact, partial immunity to herpes viruses.

The results presented in this paper raise serious question about such an interpretation. The data presented in Table 2 suggest strongly that the resistance of the rabbit eye to herpes simplex virus is strain specific. An earlier publication has pointed to antigenic differences between diffrent strains of this virus[8] in addition to the well established differences in tropism, and virulence for different animals or tissues.[7] The present work tends to lend support to the existence of such differences. Strain PET is known to differ significantly from the standard reference strain HF.[8] Strain H51 which was employed because of its known lack of encephalitogenic properties and its high virulence for the cornea, resembles HF but seems to differ from PET. Each of the strains appears to give significant strain specific resistance of the rabbit eye, without significant cross resistance between PET and H51.

The present work unfortunately did not resolve the nature of the ocular resistance. As far as defined up to the present time, its characteristics appear to be: (a) at least partial independence of circulating specific antibody titer, (b) strain specificity, (c)

at least partial independence of nonspecific factors which might tend to reduce the inflammatory response to *all* insults, as witnessed by full susceptibility to challenge by an unrelated agent like vaccinia virus. These characteristics point to antibodylike substances playing a role.

The avascularity of the cornea often has been held responsible for the lack of correlation between circulating antibody and corneal susceptibility to infection. Recent workers[10] question this interpretation because they observe capillary invasion into the corneal stroma within six hours of trauma. However, the blood vessels coursed in the substantia propria, just beneath the epithelium and thus may be compared to the subepithelial blood supply in skin which does not prevent recurrent herpetic skin lesions in the presence of circulating antibody.

The evidence presented in this paper suggests that the strain-specific resistance, which is not a direct function of circulating antibody yet seems associated with properties suggesting an antibodylike substance, may be bound to corneal cells. Perhaps it may be elaborated by corneal tissue like antibodies to chemically pure antigens.[2] Studies are contemplated to pursue these suggestions.

Summary

Systemic immunity associated with circulating antibody failed to protect rabbits against corneal infection with herpes simplex virus. The "virulence" of a strain of this virus for the rabbit cornea was unrelated to its virulence for mice.

Corneal infection with this virus gave some degree of resistance to re-infection by the same route. This corneal resistance was unrelated to circulating antibody and appeared to be strongly strain specific.

Infection of the cornea with a given strain of herpes simplex virus gave marked protection to challenge by the homologous strain but no significant protection to challenge by an antigenically slightly different strain of the same virus. The possible nature of this corneal resistance is discussed.

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THE OPHTHALMOLOGIST LOOKS AT THE READING PROBLEM*

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The purpose of this paper is to reacquaint the ophthalmologist with the problem of the child with retarded reading ability. The observations in this report are based on the study of 100 children with reading difficulties and a review of the literature on the subject (table 1).

TABLE 1

SUMMARY OF CASES

Causes of Referral	Percent
1. Educational difficulty	30
2. Aggression	23
3. Truancy	13
4. Psychological disturbances	27
5. Eneuresis	7

Within the last five years this has become at pressing question, and more has been written about it than almost any other subject. Two UNESCO meetings and a State Department conference have been held on this subject, and there are animated discussions of the problem whenever educators meet.[1] No satisfactory plan to deal with this still growing problem of the educational system has yet been presented, although there is an incidence of 20 to 40 percent deficiency[2] noted each year in the school population. This indicates that there are 8,000,000 retarded readers in our educational system. Harrower[3] points out that 75 percent of delinquent children in New York City are poor readers. Hallgren[4] indicates a definite hereditary factor in reading retardation. The mental health significance of this is well recognized. Reading difficulties are frequently found among adolescents with chronic antisocial behavior.[5] This is well recognized by law enforcement agencies.[6] The ophthalmologist, with a knowledge of the problems involved, should serve as the balance and guide in this chaotic state.

Despite the numerous etiologic factors which have been incriminated, little effort has been made to correct the condition. There are some four million first-grade pupils[7] entering the school system each year, but corrective treatment is undertaken only when the deficiency manifests itself in the form of failure in upper grades. Unless corrected the need for remedial classes and clinics will soon reach such proportions that training classes for defective readers will form a separate and important subdivision of the departments of education.

In 1843 Lordat described his own inability to read as a result of a vascular accident. Since then the problem of reading deficiency has been a perplexing one to physician, educator, and parent. There is no one etiologic factor. Children fail to read for a number of reasons, and in each case it is necessary to determine individually the causative factors. The words[8] "strephosymbolia, congenital dyslexia, bradylexia, specific dyslexia, congenital word blindness, visual agnosia" are among some of the many terms that have been used to describe the syndrome.

The characteristics of a patient with a reading retardation problem may be summarized (table 2) as follows:

He usually is a male patient[9] of normal or superior intelligence.

TABLE 2

READING DEFICIENCIES FOUND MOST COMMONLY AMONG FIRST-GRADE PUPILS

1. Inability to work out a pronunciation of a strange word.
2. Failure to see likenesses and differences in forms of words: on—no; pug—bud.
3. Failure to hear differences in sounds of letters.
4. Making reversals: u—n; stop—tops; was—saw.
5. Failure to keep the place.
6. Failure to read from left to right.
7. Vocalizing of words.
8. Failure to read with sufficient understanding.

* From The Wilmer Institute of The Johns Hopkins Hospital.

There is a surprisingly high incidence of ambidexterity or left handedness.

There is a persistence of a normal childhood tendency to reverse letters and symbols —as "p" for "q"; a tendency to reverse words, as "stop" for "pots"; in rarer cases there is actual mirror writing.

The child may later present an emotional problem, reading two or more grades below grade level but often doing better than average work in arithmetic.

The slow reader must be differentiated from the retarded reader. A slow reader is one who reads below grade level but this reading level is consistent with his intelligence level. A retarded reader is one who reads below grade level but has a normal or high intelligence quotient. The slow reader will not be considered further in this paper and constitutes a separate clinical problem.

A modification of an excellent study by Rabinovitch[10] divides the retarded readers into two major groups:

I. (a) Those in whom reading retardation is due to frank brain damage. An example of this would be the cerebral palsy child. (b) Those in whom the defect seems to be a basic incapacity to integrate written material and to associate concepts with symbols. This is primary reading retardation. Here one is dealing with a subclinical brain injury or a developmental discrepancy. Although the primary reading-retarded group shows no gross neurologic dysfunction, there are motor and sensory changes that can be detected by more exact qualitative examinations. Rabinovitch feels that the parietal-occipital areas show the primary dysfunction. These children, as distinct from those with secondary reading retardation, are difficult to retrain.

II. Those who have poor reading achievement but normal intelligence and no defects in basic learning capacity. These are due to exogenous factors and are called secondary reading retardation. One patient in this group was of special interest. She was a child of seven years, referred from one of the schools because the teacher felt she was handicapped and should be retained in the first grade for another year. Under proper psychiatric guidance, in four months she was able to improve her reading achievement equivalent to 15 months.

On each of Rabinovitch's 250 patients the following examinations were done: (1) Psychometric evaluation, (2) achievement testing, (3) psychiatric evaluation, (4) neurologic, and (5) response to remedial therapy.

The understanding of the term "reading readiness"[11] is basic to the understanding of the reading problem. Each child who begins school has a varied and different physical, emotional, and educational background; yet, by tradition, at the moment the child becomes six years of age, the parent, teacher, and society in general expect him to be ready for reading. This is not to be expected with each child!

Under ideal conditions the child is prepared to make an easy transition from oral language to written language. He learns to listen, then to talk; he learns how language is organized in the form of sentences and paragraphs. This combination of concepts, attitudes, and interests provides the foundation on which reading is built. It takes five to eight years to develop this reading readiness.

With each child there is some variation in the facility he manifests for reading readiness. At the strategic age of six years many factors might influence or retard the coordinated development of this reading skill and play a prominent role in the causation of a reading problem.

The goals leading to reading readiness should stress:

1. Social adjustment in school situations; learning to share ideas and possessions.

2. Development of basic concepts essential to everyday living and to initial reading instruction.

3. Advancing the child's comprehension and use of oral language.

4. Stimulating interest in reading.

5. Developing an awareness of relationship between language and personal experience.
6. Detecting physical and emotional needs.

The problem of the educator

There are a few basic educational terms and methods of teaching reading which must be mentioned before discussing the roles that psychiatry, education, ophthalmology, neurology, otolaryngology, and remedial reading will have to play in this complex subject.

About 1926, the method of sight reading and phrase reading was introduced by the psychologists and educators because it was faster and more efficient and gave the reader a better idea of the content of the subject matter.[12] The present educational system proposes the learning of 75 basic words by the sight, flash, or word recognition method.[13] Then, at the end of the first grade to the third grade, phonetics and structural analysis of these 75 basic words are practiced. Deviations and variations from this procedure of study are too numerous to list. They vary with the area of the country, the schools, and the teachers. The amount of phonetics taught is often completely in disproportion to the degree of sight reading.

It has become fashionable to overemphasize sight reading for all children. The teacher who taught phonetics was in danger of being designated "old fashioned" and, too frequently, when the supervisor made rounds, the phonetic chart was hidden from view. The young teacher was often ignorant of phonetics or how to teach such a subject.

Gray,[14] an educator at the University of Chicago, states that elaborate and extensive remedial reading programs in the intermediate and upper grades are evidences of failure to provide adequate developmental reading programs in the elementary grades. He further states that, during the last 20 years, teachers have had little preparation for the teaching of structural and phonetic analysis.

This emphasis on sight reading has caused students who would read well under any circumstance to progress more rapidly but the student who, because of neurologic, emotional, or other nonspecific cause,[15] was unable to learn with this method, was caught in a stream of confusion in which at least 20 to 40 percent of our student body are now hopelessly floundering.

Orton[16] claims that there are three times as many reading problems in children taught by the sight method as in those taught by the phonetic approach. Monroe,[17] who advocates a phonetic approach, states it is better to be a slow reader than a nonreader. Even though the educator has become aware of this situation, the harm has been done to both students and recently graduated teachers. Only slowly do the authorities admit the error, and too gradually does the pendulum begin to reverse itself. Instead, the educator seeks to place the responsibility on other factors, all of which may be of some importance, but none of which completely explains the difficulty. These other factors (table 3) are: (1) Overcrowded schools, (2) overworked and inexperienced teachers, (3) transient population, (4) compulsory promotion of students, and (5) compulsory education of student to the age of 16 years.

The remedial reading class,[18] too, shares in this state of confusion. There are no enforced qualifications for a remedial teacher and each teacher can develop his or her own pattern of instruction. A subject so complex as teaching at the remedial level cannot be done by the retired school teacher, or by one who is not too successful in her regular area.

Ophthalmologists[19] who have written on the subject have been too prone to blame all of the failures on the "phonetic ghost." In

TABLE 3

Educational factors in reading retardation

1. Sight or flash method of 75 basic words
2. Overcrowded schools
3. Overworked and inexperienced teachers
4. Compulsory promotion of students
5. Compulsory education of students to age 16 years
6. Transient population

this way we have estranged ourselves from the educator. As a result, in many areas the educators are seeking the more sympathetic ear of the commercial entrepreneurs. The estrangement of educator and ophthalmologist should not be allowed to exist. There is a sincere desire on the part of the educator to participate in a co-operative effort but not at the sacrifice of his own independence, nor under the constant criticism of his reading program.

Irvine[20] states that the reading problem is of importance to the ophthalmologist, for it is to him the parent and school authority turn for advice and scientific appraisal. The school authorities recognize the leadership of the ophthalmologist but he should continue to merit this trust and be ready to give advice when it is requested. Any apparent lack of unanimity in advice will cause the school authorities to heed the advice of others, who, even though tainted by commercial interest, nevertheless present a united front.

The role of ophthalmology

The ophthalmologist's role in reading disability is the same as in any systemic disease with ocular manifestations. Defective vision and muscle imbalance do not have any significant role in the etiology of a condition influenced by spatial confusion and poor visual memory.[21] If visual acuity is reduced, the child will have difficulty in interpreting symbols, just as the deaf child will have difficulty with pronunciation.

The importance of low degrees of refraction is grossly exaggerated. Muscle imbalance and strabismus do not affect the interpretation of symbols but the effort to overcome such a weakness and to see binocularly may cause fatigue and discourage reading. There may be alternate fusion and suppression. Convergence insufficiency and other muscle anomalies have been found in a large percentage of cases (Park). These defects may result in the slow reader, but have little or nothing to do with the retarded reader.

The ophthalmograph is an instrument that may demonstrate the mechanism when such fatigue results. The use of the tachistoscope and metronoscope are of importance, not in correcting an ocular defect or a muscular weakness but in providing extra motivation and training in reading when used at the remedial level. The improvement that occurs after use of such instruments is the result of this added motivation.

The role of the neurologist

Broadbent and Kerr[22] conceive reading disability to be a specific neurologic dysfunction. Bender and Schilder[23] de-emphasize localization. Morgan[24] expresses the opinion that reading disability is a developmental lag with ultimate spontaneous maturation of function. Hinshelwood,[25] in 1917, ascribed the lesion to be a failure of the cerebral cortex to develop. Orton,[26] in 1926, felt the condition was not a pathologic factor, but a physiologic deviation due to failure of acquisition of the normal pattern of complete dominance of one hemisphere of the brain. He expressed the belief that the two cerebral hemispheres received mirror images and that one becomes dominant. If the right cerebral hemisphere is dominant the individual becomes left handed, which leads to mixed dominance and exaggerated reversals.

There is strong presumptive evidence that the cases of primary reading retardation are associated with a lesion in the parietal-occipital lobe.[27] The cases of acquired alexia characterized by difficulty in spatial orientation and visual agnosia have been proved at autopsy to have pathology in this area. Lindenberg[28] states that this is the border zone supply area of the anterior, posterior, and middle cerebral arteries. In cases of hypoxia there is damage to the parietal-occipital areas. This results because the anterior cerebral artery, whose terminations lie in this area, commences as a major vessel in the region of the frontal lobe, and then, after coursing over the entire area of the brain, terminates in

the occipital area. In cases of hypoxia it is this area that first demonstrates degeneration of the ganglion cells. These border zones are the predilection areas of nerve cell necrosis. These cells have to do with spatial orientation and visual agnosia; therefore, retarded reading could develop as a result of the hypoxia that frequently occurs at birth.

Kawi and Pasamanick,[29] in a review of the prenatal and paranatal records of 205 children with reading retardation, found that 16.6 percent had been exposed to two or more maternal complications, as compared to 1.5 percent of a similar group without reading disorders. The complications were such that would lead to fetal anoxia.

Each hemisphere is adequate, since the lesion which cuts only the homolateral pathway does not cause alexia, even though it is in the master hemisphere. Each macula is connected with both cortical areas. Sherrington's flicker experiment demonstrates that functionally the two eyes are interchangeable. It would seem that it makes no difference which eye is used for sighting, since the image from either eye is related to the higher centers.

Although Klingman found that there was a retention of immature electroencephalogram patterns in 80 percent of the retarded readers, and Kennard found that 70 percent of patients with retarded reading ability had abnormal electroencephalograms, these figures have not been substantiated by others. The general consensus is that the electroencephalogram pattern is not significantly abnormal in retarded readers.

Besides the gross neurologic defects associated with various forms of alexia, many patients with reading retardation problems exhibit a lack of co-ordination. This is exhibited by clumsiness in buttoning a shirt, or by poor hand-and-eye co-ordination in bouncing a ball. Gesell[30] states that an unrecognized minimal birth injury may express itself in speech difficulty and may later result in serious difficulty in the acquisition of reading.

The role of psychiatry

In our present culture, where the ability to accumulate facts is through the avenue of reading, the problem of the retarded reader has directed the concern of parent, child, and teacher to an emotional level.

There are two groups of emotionally disturbed children who may develop reading problems:[31]

I. The child who has deep emotional problems prior to the beginning of school.[32] These emotional problems may be concerned with chronic alcoholism in the parents, in divorce, in the death of one or both parents, and in any of the many other causes for such basic emotional disturbances. This child is poorly fitted for the study of the complex mechanisms involved in reading. The lack of necessary concentration will more than likely lead to confusion and eventual reading retardation.

II. The emotional disturbances which result after beginning school.[33] These may be due to a host of factors, and among them are: (a) Transient population, resulting in frequent changes of school; (b) language difficulty, as caused by varying dialects in different parts of the country; (c) auditory impairment. These are frequent factors which lead to delay in emotional security and reading readiness.

The four common types of emotional reaction associated with reading retardation may be expressed by

1. The "couldn't care less" attitude.
2. Paranoid reaction to the teacher.
3. Marked feeling of inferiority.
4. Tendency to emotional blocking, with the possible development of deeper emotional disturbances.

The introverted child may become more introverted, and frequently schizoid in type. The extroverted child may react in a belligerent manner which evidences itself in

ways varying from minor behavior problems to the more serious problems of major delinquency. A feeling manifested by both types is the utter dislike for reading, and the very sight of a book causes feelings of nausea and resentment. This feeling, in some, has become so intense that it has led to theft and attempted arson, which most often are related to the reading teacher. In any attempt at considering this underlying difficulty the first problem is to break down these antagonisms. This is the problem of the child, but the parent, also, must be considered.

The parents, alarmed, puzzled, and defensive when their child does not do well in reading, try to prove both to themselves and their neighbors that their child is not handicapped. This apprehension is reflected in the emotional state of the child and makes reading more difficult. Any assistance the parent could give is rapidly dissipated in the anger and frustration that arises between parent and child. When this friction mounts to white heat it can subside only when the teacher, or the system, is blamed. As a result of such a situation, the child is in a dilemma which he cannot understand, and from which he cannot escape. Then ensues a loss of self-confidence which may lead to hostile acts and utter rejection of the entire reading program. Unfortunately, this reaction does occur in the child with a normal or high intelligence level.

The fact that 15 percent[34] of children reaching school age are not ready for instruction in reading, since only a makeshift plan is attempted with such children, makes it clear that we are starting with a 15-percent deficiency due to this single factor of immaturity. This group of children will begin to read when they are ready to do so, provided they are protected from unnecessary competition and the stigma resulting from frustration and the disgrace of failure. This points up the necessity of co-operation between parent, child, and teacher.

Summary

There is no single proper way to teach reading. Sixty percent of the children learn to read satisfactorily by any method. Most learn to read better by a combination of methods, and with emphasis on the sight method. There are some who, by reason of physical, educational, or environmental difficulty, cannot read by the method of instruction proposed. An effort to recognize this group should be made at the beginning of school, and remedial therapy should be instituted before the problem has reached such proportions that it is almost hopeless to correct.

A child presenting with a reading problem should first be referred for psychologic evaluation (table 4). One can thus immediately determine the intelligence quotient of the patient; and by the Thematic Apperception Test, the Blacky Series, and the Rohrschach Test, one is able to evaluate the personality of the child. Once this has been done one is then in a position to direct the patient in a sensible manner. If remedial reading is indicated, he should be referred for a reading analysis in order that the areas of deficiency can be determined and treated. If the child is not suitable for remedial training, proper protection should be given him in order to prevent a sense of frustration. Such a plan

TABLE 4

COMPLETE ANALYSIS

1. Individualized I.Q.—Wechsler or Binet
2. Standardized I.Q.
3. General physical examination
   a. Neurologic
   b. Visual
   c. Auditory
   d. Psychiatric
4. Personality tests
   a. Thematic Apperception Test
   b. Blacky Series
   c. Rohrschach Test
5. Informal reading inventory
   a. Reading material at different levels
   b. Word recognition
   c. Visual and auditory discrimination
   d. Spelling inventory
   e. Associative learning test

will also lessen the burden of the teacher who should be dealing with normal students only. Visual and auditory examinations are indicated in all cases, but as a part of reading readiness.

The most important thing in the remedial reading program is to make a complete analysis of the individual and his specific needs. Adams[35] feels that a child suffering from reading disability is educationally ill, and before any remedial teaching is started each case should be diagnosed individually to determine the specific need of the patient. If there is any emotional instability in the child, this should be determined, and treatment begun, before any remedial therapy is commenced. Any attempt at prophylactic measures will get its cue from remedial teaching. Therapy for the primary retarded child should be in the form of an immediate phonetic approach rather than the sight-phonetic approach now used. This direct phonetic approach combines structural, phonetic, auditory, visual, and kinesthetic methods; that is, this approach teaches the child how the letters are put together, how they are spoken, how they sound, how they look, and how writing reinforces these stimuli.

Although most cities have well organized remedial reading centers, there is none with a logical educational program in the early teaching of reading skills which is designed to obviate the pitfalls which make the remedial reading centers necessary.[36] With early detection much preventative work can be done, and therapy will not be as difficult then as it is in the later grades, when there will be associated secondary emotional problems.

Conclusions

1. A review of 100 cases of reading retardation confirmed Rabinovitch's classification of primary and secondary reading retardation. A possible etiologic factor was indicated in every case.

2. Refractive errors and muscle imbalance play only a small part in the problem of the retarded reader.

3. It is possible to recognize a reading-retarded child at the first-grade level. It is essential that we deal with the problem at this level before the development of secondary emotional complications.

4. It is necessary that we work, not as individuals but as a team, in an all-out effort to defeat this difficult educational problem.

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READING FAILURES AND NONFAILURES*

In children with brain damage

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The child with brain damage is generally believed to exhibit various physical handicaps together with some degree of inability to learn and retain material taught in the classroom.[1-3]

The present investigation was made to find what ocular conditions, if any, are characteristic of or occur more frequently among these children, especially among those who fail in learning to read. It is a part of a long-term study of physical handicaps which interfere with learning, a number of the ophthalmologic reports of which have appeared in this magazine.

The case records of 2,400 school children between the ages of five and 17 years were taken from my files and reviewed for the presence of brain damage. Of these children, 1,000 were not failing in their school work and 1,450 had been referred because of difficulty in learning to read. Diagnoses of brain damage were in 1.4 percent of the entire group, while the figure for the nonfailing group was 1.1 percent and for the reading failures 1.6 percent.

The entire group of cases of brain damage presented a median age of 10 years, median grade of third, median birth weight of 7.1 lb., median IQ of 90, and median age of appearance of damage four years. This contrasts somewhat with the medians of the nonfailing group—age, nine years; grade, fourth; birth weight, 6.6 lb.; IQ 90; age of onset, 7.6 years. The failing group paralleled the total group of cases of brain damage somewhat more closely, with medians of age, 10 years; grade, third; birth weight, 7.6 lbs.; IQ 90; age of onset. 3.6 years.

It should be noted that the nonfailures presented a younger median age and that

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their older median age of onset implies that a larger proportion of them suffered the brain damage after school entrance and so had some opportunity to adapt themselves to the school as relatively normal children. The majority of the failures suffered their brain damage before entering school and were, therefore, at some disadvantage. The somewhat lower median birth weight of the nonfailing group seems to be of no particular significance since it is within the limits of normal.

Comparisons were made between the total group of children with brain damage and an unselected group of 500 cases—between the nonfailing and failing cases of brain damage; between the nonfailing and failing groups in which the brain damage was due to application of external force; and between the nonfailing and failing groups in which the brain damage was due to defect or disease.

Categories in comparisons which follow are those displaying differences of 15 percent or more and include amblyopia, hypermetropia, muscular imbalance, strabismus, retarded speed of perception, fusion deficiency, combinations of eyedness and handedness other than right eyedness with right handedness, mental retardation, and emotional disturbances.

Visual acuity measurements were made with corrections. Cases in which glasses failed to raise vision to normal (20/20) were deemed amblyopic. The commonest type of muscular imbalance was exophoria at the reading distance but there was no predominant type of strabismus.

The total group with brain damage exhibited 17 percent more amblyopia than the unselected cases; 26 percent more hypermetropia of 1.0D. or more in the right eye and 12 percent more in the left eye; 24 percent more muscular imbalance of six or more prism diopters at the reading distance, chiefly exophoria; 19 percent more strabismus; 11 percent more mental retardation; and 25 percent more emotional disturbance.

Next the children with brain damage who were failing in reading were compared with the nonfailing cases of brain damage. The former displayed 26 percent more muscular imbalance of six or more prism diopters at the reading distance and 20 percent more lateral dominance variations, other than right eyedness with right handedness.

Cases of brain damage due to external force were compared as to reading failure and nonfailure. Concussion was the most prominent feature in the histories of these patients. Twenty percent more of the reading failures exhibited semantic aphasia (word blindness) and 50 percent more amblyopia, while 17 percent of the nonfailing group presented fusion deficiency and 16 percent more lateral dominance conditions other than right eyedness with right handedness.

A similar comparison was made for the cases of brain damage due to disease and defects. Meningitis and encephalitis were prominent in this group of case histories. The reading failures presented 77 percent more muscular imbalance of six or more prism diopters at the reading distance, 21 percent more strabismus, and 15 percent more retarded speed of perception, while the nonfailures exhibited 36 percent more amblyopia and 49 percent more fusion deficiency.

The chief types of reading errors made by those cases of brain damage in the reading failure group included poor comprehension of what was read in 42 percent; trouble with word endings in 21 percent; poor recall and poor mechanics of reading, 15 percent each. The children with brain damage due to diseases and defects appeared to make about the same types of reading errors as those children with brain damage who failed in reading; however, the children with brain damage due to external force tended to be unable to recognize words until they were spelled or written. This implies that the injury had impaired the visual-symbolic pathways in the brain (Brodmann areas 17, 18, 19, and 39), making necessary oral and kinesthetic reenforcement.

Various investigators[3-9] have reported higher frequencies of amblyopia, hypermetropia, muscular imbalance at the reading dis-

tance, fusion deficiency, retarded speed of perception, and lateral dominance variations among reading failures. The present study also shows a higher frequency of these conditions in some of the cases of brain damage failing in reading, which raises the question whether it was the brain damage or the reading failure that came first and, perhaps, produced them.

The figures show that the majority of the reading failures with brain damage suffered the damage before entering school, which would make it a more likely cause of reading failure than possible emotional reactions. Those with a medical background would tend to attribute these conditions to pathologic influences and to regard the brain damage as a probable cause. They might also consider subclinical brain damage to be one of the causes of poor reading among those children who had not been diagnosed as having brain damage. The person with psycho-educational training might concede to this medical view but he would also point out that reading failure could produce the eye conditions, or some of them, through emotional reactions to long-continued school failure.

The fact that the nonfailing cases of brain damage exhibited more amblyopia and markedly higher frequencies of hypermetropia than the unselected cases suggests that these conditions are more fundamental than the emotional difficulties arising from reading failure, especially since none of the members of the group were failing in any subject. The higher frequency of emotional disturbance among the nonfailing cases of brain damage, as compared with the unselected cases, lends support to the idea that brain damage results in more frequent departure from stable emotional conditions.

Fusional deficiencies, although sometimes regarded as factors contributing to difficulty in learning to read, have never shown markedly higher frequencies among poor readers than among the unselected, although their incidence is a little higher. In the present study the nonfailing children with brain damage exhibited five percent more cases of fusion deficiency than the unselected cases. While this difference is too small to be regarded as more than a suggestion of a trend, it implies that brain damage tends more frequently to accompany fusion deficiency.

This study is limited by the small number of cases of brain damage and each successive subdivision and comparison, with its correspondingly reduced population, is necessarily more limited. However, to the extent that the groups are representative, it appears that:

1. Certain eye conditions are more common among children with brain damage than among those without it. These conditions include hypermetropia of 1.0D. or more, amblyopia, muscular imbalance of six prism diopters or more *at the reading distance*, and strabismus, as well as emotional disturbances.

2. Children with brain damage who fail in learning to read tend to exhibit more muscular imbalance of six or more prism diopters at the reading distance and more lateral dominance variations than children with similiarly damaged brains who have no difficulty in learning to read.

3. Children with brain damage due to external force who fail in reading tend to exhibit more semantic aphasia, amblyopia, and emotional disturbance; while those with brain damage who are not failing display more fusion deficiency and lateral dominance variations.

4. When the damage is due to disease or defect, the tendency is for the reading failures with brain damage to present more cases of muscular imbalance of six prism diopters or more at the reading distance, strabismus, and retarded speed of perception, while nonfailures with brain damage show greater frequency of amblyopia and fusion defects.

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METASTATIC DISEASE OF THE OPTIC NERVE*

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Metastatic disease of the optic nerve as an extension of an ocular tumor is frequent in retinoblastoma, not common in malignant melanoma of the uvea. Secondary involvement of the optic nerve by continuity from primary tumors of the orbit or the nasopharynx will not be included in this study, only true (embolic) metastases will be considered.

Duke-Elder[1] lists 12 cases of metastatic disease of the optic nerve due to carcinoma. In most of these 12 cases, the secondary growth was located in the sheaths of the optic nerve, with involvement of the intracranial segment first and later extension into the orbital portion. Only one case of metastatic disease of the optic nerve from a primary malignant melanoma of the skin is reported; two cases were due to lymphosarcoma.

As metastases to the central nervous system are not rare, the paucity of reports in the ophthalmic literature on involvement of the optic nerve was assumed by some authors to be only apparent. They believe that such occurrence was not unusual; it merely remained undetected in patients often too ill to voice their visual difficulties.

It seemed of interest, therefore, to consult reports from neurologic and pathologic sources as well as from research on cancer on large series of metastatic disease of the central nervous system and note the incidence of involvement of the optic nerve. When the wealth of literature on this subject is reviewed (including large numbers of necropsies), one is struck by the variation in incidence of metastatic disease to the central nervous system, as given by different authors. Krasting[2] records an incidence of 4.7 percent and Lesse and Netsky,[3] 34.8 percent.

The involvement of the brain in secondary neoplastic disease is an expression of a general widespread seeding of the primary tumor. However, there is a well-known predilection of some neoplasms to metastasize to the central nervous system—carcinomas of the lung, the breast, and gastro-intestinal tract, and melanomas of the skin.

The increase of the incidence of carcinomas of the lung in the last two decades is reflected in the greater percentage of metastatic disease to the brain in the more recent reports on this subject. The completeness of the brain examination (including microscopic studies) in the newer literature is, of course, another factor upgrading this incidence.

Melanomas account for about one percent of all malignant tumors. They metastasize in in about 100 percent of cases and, according to Bailey,[4] about 50 percent of them metas-

* Read in part before the North Carolina State Medical Society, Ophthalmology Section, Asheville, May 6, 1957.

tasize to the central nervous system. Krasting, Ewing, and Rasmussen and Kernohan and Adson of the Mayo Clinic believe that most cerebral metastases in melanomas arise from a primary tumor in the skin.

The literature[5-22] of 1,595 cases with autopsies of metastatic disease of the central nervous system, including microscopic studies in a majority of instances, was reviewed. In 212 of these cases the primary tumor was a malignant melanoma. The other cases were carcinomas with a very few exceptions (Grawitz tumor, and so forth).

Though many of the histories had notations on visual impairment, the necropsies only partially bore out this finding. The occipital lobes were the seat of metastatic growth in a considerable number of these cases. Invasion of the optic thalami and the optic radiation was not so frequent. In only four cases was the intracranial portion of the optic nerve involved; in one case the orbital segment of the optic nerve.

Sometimes the blindness existing before death could not be accounted for by the objective findings at autopsy and some authors assumed that toxins liberated from the neoplasm was its cause. More recent investigators are not in accord with such an hypothesis and believe that microscopic tumor emboli to the visual centers and pathways account for such an occurrence. In the four cases with involvement of the intracranial portion of the optic nerves, the leptomeninges were diffusely infiltrated and other cranial nerves were also affected (table 1).

The clinical diagnosis of metastatic disease of the optic nerve is difficult for the symptoms vary with the part of the nerve involved. A metastatic focus in the intraocular portion of the optic nerve is visible ophthalmoscopically as a new growth of the optic disc. Metastases in the orbital part of the nerve present only symptoms of visual impairment, as in retrobulbar neuritis. If the intracranial segment of the optic nerve is involved, the ophthalmoscopic examination may be negative, or there may be papilledema due to an increase of the intracranial pressure subsequent to other metastases in the cranium.

According to Abelsdorff,[23] the visual impairment is greater if the intracranial section of the optic nerve is involved. The field defect is not characteristic unless the metastasis has involved the chiasm. Pallor of the optic disc is rarely noted even in cases of amaurosis. He believes that the initial lesion to the intracranial portion of the optic nerve occurs in its sheaths, which are rich in blood vessels, with resulting compression of the nerve followed by invasion and destruction of the nerve substance.

Besides the optic nerve other cranial nerves may be involved as part of a general seeding to the central nervous system and such symptoms of meningeal or brain irritation as headaches, dizziness, nausea, and vomiting are frequent. A history of a tumor elsewhere will assist in the diagnosis, though sometimes metastases to the central nervous system may appear before the primary malignancy is disclosed—or the primary site cannot be found.

Case report

A 42-year old white woman noted loss of hearing and impairment of vision on the right side two weeks prior to admission to the hospital. Vertigo, nausea, and vomiting developed soon afterward.

Her past history revealed diphtheria, pneumonia, appendectomy, and tonsillectomy. She was allergic to many foods, tobacco, dust, and so forth, for which she received desensitizing injections. Her mother had died of carcinoma of the breast, otherwise her family history was noncontributory.

At the physical examination heart and lungs were found to be normal, there were no masses in the abdomen. Blood pressure was 166/110 mm. Hg. Impairment of vision and loss of hearing on the right side were present. The admitting diagnosis was cerebral hemorrhage.

The ophthalmologic examination revealed both eyes to be externally normal. Pupils were round, equal, with good light reaction. No nystagmus or diplopia was present.

Fundus examination showed the disc of both eyes to be of normal color and outline. The retinal arteries were somewhat narrower than normal. No hemorrhages, exudates, or pigmentations were present anywhere in the fundus. Corneal sensitivity was normal and equal on both sides. Vision was: R.E., 20/25; L.E., 20/20.

TABLE 1

CASES OF METASTATIC DISEASE OF OPTIC NERVE TAKEN FROM PATHOLOGIC AND NEUROLOGIC SOURCES

Name	Number of Cases	Optic Nerve Involvement	Primary Tumor	Involvement of Brain
Krasting[2]	1	Left, intracranial	Carcinoma of vagina	Nodule in dura
Mayer[7]				
Case 4	2	Intracranial portion	Undetermined	Leptomeninges diffusely infiltrated
Case 5				Calcarine cortex invaded
Globus and Selinsky[10]	1	Intracranial portion, left side	Carcinoma of bronchus	III, VIII, & X, and leptomeninges
Bork[11]	1	Orbital portion	Carcinoma of breast	Nodule in dura at base of brain

Field studies of the right eye showed an irregular defect for white and colors in the nasal-upper quadrant reaching to the fixation point. The field of the left eye was normal. The diagnosis was a lesion of the right optic nerve posterior to the globe and anterior to the chiasm.

Laboratory findings showed serology to be negative, as were blood examinations and urinalysis. The spinal fluid was clear, under no increase of pressure; total proteins 73 percent, 20 cells, with 78 percent of lymphocytes. X-ray studies of the skull were negative.

Under sedatives and bedrest, nausea, vomiting, and vertigo disappeared. The blood-pressure became lower and she left the hospital four days after admission. A week later she was readmitted because of severe headaches and ataxia. A cervical adenopathy was noted, the chest X-ray film showed enlarged mediastinal glands. Ataxia and a positive Romberg were present, otherwise the neurologic examination was negative.

The following day a moderate stiffness of the neck developed and a weakness of the right facial nerve with some lagophthalmos. The right pupil was enlarged, light reaction sluggish and incomplete. The fundus was entirely normal, vision was reduced to seeing hand movements with the right eye. The vision of the left eye and field were normal.

A cervical lymph node was removed and the pathologic diagnosis was metastatic tumor. A clinical search for the primary tumor was unsuccessful; then the family volunteered the information that four years earlier a reddish mole had been removed from the skin of patient's back. Inspection of the site of the former skin lesion showed a very faint scar; the surgeon who did the operation in his office termed it a benign nevus. No microscopic examination had been made.

The course of her illness was progressively downhill, aggrevated by much pain in every part of her body. Soon an exposure keratitis developed in the right eye. Four weeks later a beginning papilledema of the left optic disc was noted, followed shortly by papilledema of the right optic disc. A few days before death she became deaf in the left ear and she expressed concern about her vision in the left eye, which was reduced to 20/70. Because of her confused mental state, the field could not be tested. She died two months after onset of her illness.

At autopsy, no nevi or other lesions were noted in the skin of her body, scalp, or nailbeds. Small gray, tan, or hemorrhagic nodules were present in the subcutaneous tissues. Similar nodules existed in the thyroid, breast, peritoneum, pancreas, ovary, supraclavicular, mediastinal, and peritoneal lymph nodes. The liver, spleen, lungs, and kidneys were free of tumor.

The superficial vessels of the brain were congested. At the base of the brain the right optic nerve was encased in pinkish-tan neoplastic tissue, one mm. from the chiasm to its entrance into the optic foramen. After removal of the roof of the foramen, neoplastic infiltration of the entire length of the nerve to near the globe was noted. The left optic nerve appeared grossly uninvolved.

Plaques of tumor tissue, about one cm. in diameter, were noted where pons, cerebrum, and medulla adjoin, impinging upon the orifices of the auditory nerves and seemingly growing into them. Smaller plaques of neoplastic tissue were visible on the cerebellum. In the right frontal lobe about two cm. from the surface a soft tannish-red nodule of tumor tissue about one cm. in diameter was present. The ventricular system was unobstructed. There was no evidence of primary tumor in the nasal sinuses or nasopharynx.

PATHOLOGIC STUDY

Microscopic examination showed that the tumor tissue of sections of the various organs and of the central nervous system was composed of moderate sized cells with pink-

Fig. 1 (Weizenblatt). Photomicrograph of intracranial portion of right optic nerve. Neoplastic infiltration with melanoma of its sheath and complete destruction of nerve substance. (A) Hemorrhages. (B) Necrosis. (×20.)

staining cytoplasm and round or oval nuclei containing chromatin clumps and prominent nucleoli. A few multinucleated giant tumor cells were encountered; occasional mitoses were present. The cells were arranged in cords or sheets without any particular type of structure. Areas of necrosis and hemorrhages were frequent in the neoplastic nodules.

Sparse granular golden brown pigment was noted in the cytoplasm of the tumor cells

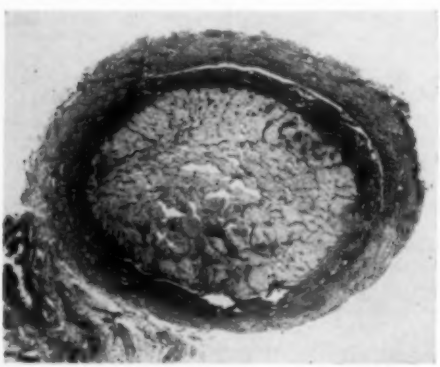

Fig. 2 (Weizenblatt). Photomicrograph of right optic nerve near optic foramen. Pia-arachnoid thickened by neoplastic infiltration. Septa of optic nerve visible, medullary sheath present at some optic nerve fibers. (×20.)

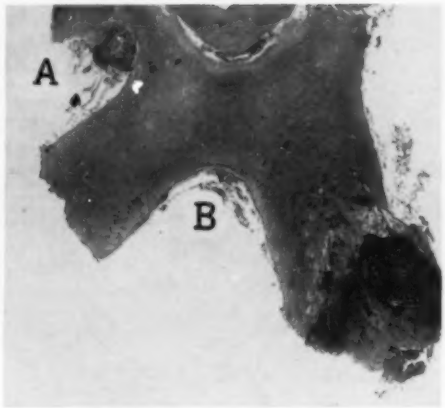

Fig. 3 (Weizenblatt). Photograph of optic chiasm, frontal section. Neoplastic infiltration, necrosis, and hemorrhages of thickened right optic nerve, one mm. short of decussation. (A) Nodule of metastatic tumor adjoining chiasm. (B) Infiltration of leptomeninges. (×6.)

in many sections. The pigment in the neoplastic cells did not stain positively for iron and it could be bleached; therefore, it was considered to be melanin.

The meninges adjacent to the tumor nodule in the cortex of the right frontal lobe were thickened with a few lymphocytes and macrophages, as well as by congestion of blood vessels. The leptomeninges near the pons contained a few tumor cells. In the cerebellum, neoplastic cells were growing from the surface into the parenchyma. A section of the medulla including the auditory nerve on the right side showed neoplastic growths and hemorrhages.

The intracranial portion of the right optic nerve was of normal thickness. Its sheath and substance were densely infiltrated with tumor cells and hemorrhages and necroses were present (figs. 1, 2, and 3). Myelin stain revealed the complete destruction of the nerve fibers. No reactive growth of glia was noted. At the chiasm the right optic nerve had lost its normal structure. It appeared moth eaten due to a massive infiltration of tumor tissue with diffuse hemorrhage and necrosis. Only on the mesial side did a few

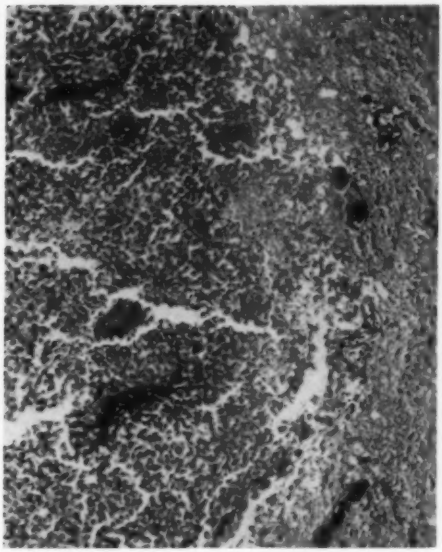

Fig. 4 (Weizenblatt). Photomicrograph of tumor nodule of Figure 3 at (A). Tumor cells in Robin-Virchow spaces in adjoining chiasm. (×100.)

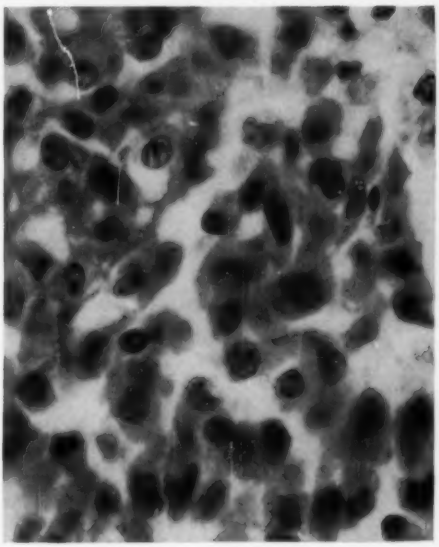

Fig. 5 (Weizenblatt). Photomicrograph, higher magnification of neoplastic cells composing metastatic nodule of Figure 4, with vesicular nucleus, prominent nucleolus, and pigment in some of them. (×850.)

nerve fibers appear intact on myelin stain. On the left side of the waist of the chiasm a tumor nodule, one cm. in diameter, was in contact with its substance and early invasion of the chiasm along the Virchow-Robin spaces was present (figs. 4, 5, and 6). The meninges of the basal side of the chiasm were infiltrated with tumor cells.

The intracranial portion of the left optic nerve showed a neoplastic infiltration of its sheath and a beginning invasion of its substance. Myelin stain showed the nerve fibers to be intact. In the region of the optic foramen the left optic nerve was free of tumor.

The diagnosis was widespread seeding of a malignant melanoma, which included the central nervous system, the optic nerves, chiasm, the facial and auditory nerves. The primary site was unknown.

Comment

Visual and auditory impairment on the right side were among the first symptoms of metastatic disease to the central nervous system in this 42-year-old woman, who died two months later. The rapidity of ensuing blindness on the right side in the presence of normal funduscopic findings seemed remarkable.

Fig. 6 (Weizenblatt). Photograph of optic chiasm. (Myelin stain, Lilly.) Complete absence of myelin of right optic nerve fibers. (×6.)

The sudden onset of her symptoms, negative physical examination, and a moderately elevated blood pressure prompted the diagnosis of a cerebral vascular insult. Two weeks later a cervical adenopathy was noted and a biopsy of a lymph gland revealed metastatic tumor; a search for the primary tumor proved fruitless.

At autopsy generalized seeding of a malignant melanoma was noted. The absence of secondary neoplastic invasion of lung, liver, and kidneys was unusual. In the central nervous system the multiple involvement of cranial nerves (II, VII, and VIII) was remarkable, as the leptomeninges were not diffusely affected. (In primary malignant melanosis of the leptomeninges, however, the cranial nerves at the base of the brain are regularly implicated).

The site of the primary tumor was unknown. However, in view of the frequency of metastatic involvement of the central nervous system due to primary melanoma of the skin and the history of removal of a mole from the skin of this patient four years prior to death, such an origin could be considered. Willis[22] describes the behavior of melanomas as: "A 'mole' unsuspected of malignancy or so small as to be overlooked may produce widespread fatal metastases."

In the majority of cases of metastatic disease of the optic nerve recorded in the ophthalmic literature and in the five cases from pathologic and neurologic sources reported here, a diffuse metastatic involvement of the leptomeninges existed and other cranial nerves were also affected. Only in Case 5 (table 1) was the orbital portion of the optic nerve involved. No case of metastases to the optic nerve was found in the 212 cases of malignant melanoma with secondary growth to the central nervous system.

The incidence of involvement of the different parts of the brain in metastatic disease is in direct relation to their bulk and vascular supply; no wonder then, that such a small structure as the optic nerve is not so often implicated as the occipital lobes and other cerebral structures.

Summary

The representative literature on metastatic disease of the central nervous system was reviewed to determine the occurrence of optic-nerve involvement.

It was found that clinically observed impairment of vision or blindness in these patients is frequently due to metastatic invasion of the occipital lobes or other visual centers; only rarely was the optic nerve the seat of a metastasis.

The five cases of metastatic involvement of optic nerve found in this study were added to those cases listed in the ophthalmic literature, in all of which the primary tumor was a carcinoma.

A case of metastatic involvement of the intracranial segment of the optic nerves due to a melanoma in a 42-year-old white woman is reported. The site of the primary tumor is unknown.

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PERCUTANEOUS DIVISION OF THE EXTERNAL CANTHAL LIGAMENT

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INTRODUCTION

With the widespread use of such improved drugs as sedatives, analgesics, muscle relaxants, tranquilizers, local anesthetics, and so forth, as well as hyaluronidase and the curarelike drugs, today few cataract extractions require external canthotomy. The usually practiced technique of dividing all layers at the external canthus followed by division of the ligament itself is a very satisfactory procedure, easily and quickly performed. Whether or not the wound is sutured, healing is usually prompt and without noticeable scar. However, I have found a few disadvantages to the procedure; this paper will suggest a different approach to the problem.

HISTORICAL

In 1839 Von Ammon[1,2] described his canthoplasty (canthotomy); apparently, however, he did not use this in ordinary cataract operations. Fuchs[3] listed the following indications for canthotomy: (1) Blepharophimosis and ankyloblepharon, (2) blepharospasm, (3) gonorrheal conjunctivitis, and (4) a preliminary step in removing an enlarged eyeball, or an orbital tumor.

Beard[1] mentions additional indications, such as: preliminary to exenteration; or extraction of cataract where the conjunctival sac is much shrunken; to relieve pressure, as in phlegmon of the orbit; and in panophthalmitis. According to deSchweinitz,[4] Axenfeld advised the procedure prior to cataract extraction in cases where the palpebral fissure is very narrow. It has long been used as a step in many types of plastic operations.

Chesshire,[5] in 1872, reported his use of a simple cut at the external canthus as a prelude to cataract surgery but he apparently did

not divide the external canthal ligament. Experience shows that little effect is obtained by this simple cut. However, a great release of tension follows division of the ligament, a fact that was noted by Vannas.[6] A consideration of the anatomy of the region readily explains why this is true. As long as the orbicularis can pull against an anchor point (lateral orbital tubercle) posterior to the apex of the cornea, it can conceivably exert pressure against the eyeball.

Vannas[6] described a procedure utilizing somewhat the same principle as in the method to be described, but he used it for a different purpose. He divided the external canthal ligament and orbicularis muscle after splitting the lids in the external canthal region in such a manner that the resulting wound was along the muco-cutaneous border, without any actual incision in the skin or into the conjunctiva of the lateral fornix.

Complications of the usual canthotomy

Hemorrhage

Case 1. In an 82-year-old woman, vertical mattress sutures were used to close the canthotomy and check hemorrhage at the conclusion of cataract surgery. After the patient had been returned to her room, she began to bleed rather briskly. Five hours after surgery, additional mattress sutures were placed and tightly tied. Hemorrhage persisted in spite of this, with tremendous clots in the upper and lower fornices and, about nine hours after surgery, the patient was taken back to the operating room. The wound was completely reopened. Two very superficial bleeders, representing the cut ends of the inferior lateral palpebral artery,[7] were clamped and tied. Apparently no harm was done to the eye in this case and the eventual outcome was good. No cause other than arteriosclerosis was found for this hemorrhage.

Case 2. A 69-year-old woman bled profusely from her canthotomy wound, yet no localized bleeding point could ever be found. A rather severe hematoma of the orbit ensued but the eye healed without any intraocular hemorrhage or other difficulty. Postoperatively, the prothrombin time was 13 seconds against a control of 18 seconds. The platelet count was 76,000 after surgery and was 114,000 on the second postoperative day. About 30 years previously, she had been advised against tonsillectomy for fear of hemorrhage.

Case 3. A 75-year-old man had a fairly severe orbital hematoma without visible harm to the eye. Aside from age and arteriosclerosis, he suffered from severe coronary insufficiency. His external canthal ligament was very tough and the extra cuts needed to divide it may have contributed to the hematoma.

Symblepharon

In Case 3, a symblepharon formed between the external canthus and the globe, causing the patient an uncomfortable sensation when he turned the eye to the opposite side. This condition was bothersome enough to require surgical correction, which was done without difficulty.

Disturbed lid mechanics

In a patient, aged 88 years, the canthotomy wound did not heal in perfect apposition. Also, there was considerable residual relaxation of the upper lid. These two factors permitted the upper lid to close completely over the lower lid. This was easily corrected with a Fuchs tarsorrhaphy.

Blepharospasm

Finally, it is my impressoin that in some of these cases manipulation of the eyelids during postoperative dressings tends to reopen the wound and causes the patient discomfort, resulting in transient blepharospasm.

Discussion

Since the main source of the hemorrhage is very superficial and near the external canthus, and since the greatest effect of the canthotomy comes from the division of the external canthal ligament alone, it was decided that there must be a better method of achieving the desired result. This idea is so obvious that no doubt others have already used it, but I have found no record of it in the literature.

The method to be described avoids cutting the inferior lateral palpebral artery, thus minimizing hemorrhaging. It avoids cutting the conjunctiva, thus preventing symblepharon. Manipulation of the lids postoperatively does not tend to separate the wound. If desired, the procedure can at any stage

during the operation be converted instantly to the usual canthotomy.

Procedure

After infiltration of the area with anesthetic, the skin above and below the lateral orbital tubercle is picked up with two forceps and a cut is made between them with the scissors. The lateral palpebral raphe is then grasped and pulled forward in order to put the ligament on a stretch. The ligament is palpated with the scissors in the wound and cut close to the orbital margin. If desired, it can be cut under direct visualization. Several snips may be necessary to free it entirely, after which the external canthus "snaps" to a position about 10 to 11 mm. anterior to its previously firmly anchored one. A moderate-sized pair of scissors is needed for division of the ligament because it is quite tough, particularly in the older age group. After the external canthus has been displaced forward by division of the ligament, practically all pressure on the globe by the orbicularis is eliminated. The skin wound is usually closed with one stitch just before the eye is dressed.

Precautions

Since the need for canthotomy in cataract surgery is now infrequent, not enough cases have been done by the described technique to give it unqualified endorsement (six cases since May, 1954). In most cases measurements of the position of the external canthus in relation to the lateral orbital tubercle before and after the division of the ligament have been made. Prior to its severance, the canthus is 11 to 12 mm. anterior and afterward it is 20 to 22 mm. anterior to the lateral orbital tubercle. Therefore, it is obvious that, in exophthalmos greater than 20 mm., a complete effect will not be achieved and the usual canthotomy should be used. Even so, if the patient is tense and excitable after adequate sedation and anesthesia, he may be able to exert enough pressure against the globe with the orbicularis to cause vitreous loss. In such a case general anesthesia is very desirable.

Measurements of width of opening of the speculum before and after the procedure were also made. In most cases, less than one mm. difference was found. Therefore, when greater exposure is desired, the usual canthotomy must be used.

Conclusion

A brief review of the subject of canthotomy has been presented. Some complications of the usual canthotomy have been outlined. A method of dividing the external canthal ligament without involving the palpebral fissure itself, or the conjunctiva, has been described. Its advantages and limitations have been outlined.

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Addendum

In a recent cataract extraction, the procedure was easily accomplished after the section, leaving the speculum in place. It is planned to try it for the relief of symptoms in certain cases of exophthalmos.

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NOTES, CASES, INSTRUMENTS

A PORTABLE STERILIZER*

For the Application of Ethylene Oxide Gas in Ophthalmology

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A recent study in which I participated† showed the potential usefulness of ethylene-oxide vapor as a sterilizing agent. An ordinary steam sterilizer was modified so that a mixture of ethylene oxide and dichlorodifluoromethane could be vaporized in its interior. A four-hour exposure to ethylene-oxide vapor at room temperature inactivated all the organisms tested. Plastics, rubber, leather, and electric equipment were not damaged by the treatment. Complex instruments such as ophthalmoscopes, Berman locators, tonometers, and magnets were sterilized without harm.

As an extension to this work in order to make the application of this sterlizing agent more practical, a new portable sterlizer‡ which employs a solution of ethylene oxide and trichloromonofluoromethane was similarly tested. This new small unit is essentially a cylinder three-inches in diameter and nine-inches high (fig. 1), which is covered by a second cylinder in such a way that a reasonably gas-tight chamber is formed. The ethylene-oxide solution is provided in a sealed metal container. The sterilizer provides a means for admitting the contents of the container to the base of the inner cylinder where the solution evaporates, filling the chamber with vapor. At the end of the sterilization period, the vapor contents are passed through an adsorbent before the chamber is opened to minimize the operator's exposure to ethylene oxide vapor.

Fig. 1 (Skeehan). Portable ethylene-oxide sterilizer. Inner cylinder (right) with attached valve mechanism to admit the ethylene-oxide solution from the small sealed container shown in the foreground.

The entire unit weighs less than four pounds. Since it requires no fixed installation and no power source, it appears to have considerable utility in the office as well as in the operating room if it could be relied upon to sterilize ophthalmic instruments. A series of tests was carried out to judge the effectiveness of ethylene-oxide gas in this smaller sterilizer.

Procedure

Instruments were contaminated by swabbing with concentrated broth suspensions of a variety of common pathogenic organisms, resistant spores, and viruses. These were exposed for varying periods of time in the Ben Venus sterilizer. The contaminated instruments were allowed to dry for one hour at room temperature. Then sterile swabs moistened with broth were rubbed over the contaminated sites and placed in tubes of ster-

* From the Ophthalmology Section, 130th Station Hospital, APO 403, New York, New York. This work was done at the Armed Forces Institute of Pathology, Ophthalmology Section, Walter Reed Army Medical Center, Washington 25, D.C.

† Skeehan, R. A., Jr., King, J. H., Jr., and Kaye, S.: Ethylene-oxide sterilization in ophthalmology. Am. J. Ophth., **42**:424-430 (Sept.) 1956.

‡ Ben Venue Sterilizer, Ben Venue Laboratories, Inc., Bedford, Ohio.

TABLE 1

INSTRUMENTS TESTED AND SITES CONTAMINATED

Instrument	Site
Ophthalmoscope	Within battery handle socket
Schiøtz tonometer	Within shaft
Hildreth hand cautery	Within battery chamber
Czapski microscope	Hand grip
Berman locator	Tip, under plastic guard
Lancaster hand magnet	Surface of magnet; cord
Transilluminator	Under socket; knurled handle
Lens for indirect ophthalmoscopy	Surface
Loupe	Lens
Eyelid speculum, Berens	Hinge
Scissors	Between blades at rivet
Needle holder	Fulcrum
Knife, Graefe	Blade
Keratome	Blade; handle

ile broth. The instruments were thereafter placed in envelopes made of bleached Kraft paper and exposed in the sterilizer for 45 and 75 minutes respectively at room temperature. After the exposure, the contaminated sites were again swabbed, the swabs placed in broth, and the broth incubated. Incubation was for 72 hours at 37°C. Control instruments were similarly handled, but not exposed to ethylene-oxide vapor. The instruments tested and the sites contaminated are shown in Table 1. The organisms employed were: spores of Bacillus subtilis, spores of the fungus Mucor, and cultures of M. Pyogenes aureus, Pseudomonas aeruginosa, and Beta hemolytic streptococci.

RESULTS

BACTERIA AND FUNGI

All control swabs were positive for the inoculated organism within 24 hours after incubation. All exposed instruments were sterile after 75 minutes of exposure in the sterilizer. No growth occurred in the broth upon incubation for 72 hours. Exposure in the sterilizer for 45 minutes, however, did not completely eliminate the Bacillus spores, nor was the covered tip of the locator sterile after the shorter exposure. A number of sharp instruments were immersed in a mixture of blood and a 18-hour culture of M. Pyogenes aureus for 10 minutes, then removed and allowed to dry at room temperature for one hour. The sharp instruments were then carefully washed, dried on a sterile towel, and placed in their original paper boxes before exposure in the sterilizer.

Sharp instruments contaminated and washed in the same way were immersed in sterile broth and shaken for five minutes, and then aliquots of the broth were plated to determine the numbers of organisms washed off in this manner while the bulk of the broth was incubated at 37°C. for seven days. Three replicate instruments gave counts of 705, 680, and 20 organisms, respectively, while all tubes showed positive growth. Even the most thorough washing with detergent and warm water did not produce sterile instruments. All instruments exposed in the sterilizer for one hour or two hours were sterile; of four instruments exposed for one-half hour, two were sterile and two were not sterile (table 2). The edge and temper of these instruments were unaffected by the exposure, nor could any tarnish or corrosion be observed.

VIRUSES

Herpes simplex virus was obtained from rabbits' eyes infected by a CFB strain in tissue culture fluid. Instruments were contaminated by handling the nictitating membrane of the eyes and wiping exudate on the instruments. Half were exposed for one hour in the ethylene-oxide sterilizer and the other half served as controls. None of the instruments were washed. Results are shown in Table 3. In the one instrument not sterilized successfully there was marked decrease in the concentration of virus.

In another series using herpes simplex, vaccinia, and infectious bovine respiratory viruses, the control and test instruments were washed thoroughly after contamination and before exposure to air or to ethylene-oxide vapor. There was complete sterilization of all instruments exposed to the gas for one hour (table 4). On several runs, all instru-

TABLE 2

EFFECT OF ETHYLENE OXIDE ON OPHTHALMIC INSTRUMENTS CONTAMINATED WITH SHEEP BLOOD AND AN 18-HOUR CULTURE OF MICROCOCCUS PYOGENES AUREUS

Temperature \ Exposure Time	One-half Hour	Control	One Hour	Control	Two Hours	Control
20° C.	1− 1+	Plate count 705	1−	Plate count 680	2−	Plate count 20
27° C.	1− 1+	Tube +	1−	Tube +	. . .	Tube +

TABLE 3

EFFECT OF ETHYLENE OXIDE ON UNWASHED VIRUS-CONTAMINATED INSTRUMENTS AFTER ONE HOUR EXPOSURE AT ROOM TEMPERATURE

Control		Exposed to ethylene oxide		
Same Day	Two Days	Same Day	Two Days	Four Days
0	++++	0	0	+
0	++++	0	0	0
0	++++	0	0	0
0	++++	0	0	0

TABLE 4

STERILIZATION OF WASHED OPHTHALMIC INSTRUMENTS BY EXPOSURE TO ETHYLENE-OXIDE VAPOR FOR ONE HOUR AFTER CONTAMINATION WITH VIRUS CULTURES

Instrument	Control			One Hour Exposure to Ethylene Oxide (all viruses)
	Herpes	Vaccinia	IBR	
Cautery tip	0	+	0	0
Tonometer shaft	++	+	+	0
Loupe	+	+	0	0
Scissors	+	0	0	0
Keratome	0	0	0	0
Knife-handle, serrations	+	0	0	0
Eyedropper, inside tip	+	0	0	0

ments exposed to the gas were sterilized in one hour. The recovery of virus from controls was variable, but some virus was found on at least one of the group of control instruments per run.

SUMMARY

Ethylene-oxide vapor sterilizes ophthalmic instruments and equipment with no damage to the physical characteristics of these items. The gas destroys bacteria and viruses within an exposure period of one hour. The new portable sterilizer is as effective as the larger device previously tested and, in addition, is more convenient, economical, and faster in operation. Plastic, electric apparatus, glass, metal, and rubber are not harmed by this method of sterilization.

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ACKNOWLEDGMENT

Appreciation is expressed to the Virus Section of the Armed Forces Institute of Pathology for the greater part of the work with viruses used in this study, and to Miss Louise Maddox of the Bacteriology Section, Walter Reed Army Hospital.

A SUCCESSFUL HUMMELSHEIM OPERATION*

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Since Hummelsheim described the operation of transplanting the tendons of the superior and inferior recti to augment the action of a paretic lateral rectus,[1] it has been modified by many surgeons including O'Connor, Gifford, Peter, and Gibson. Callahan has presented evidence that it is not the transplantation of a part of the superior and inferior recti to the insertion of the lateral rectus that accomplishes abduction of the eyeball, but rather resection of the lateral rectus combined with recession of the medial rectus which produces the result.[2]

The present case is interesting in that a pure Hummelsheim procedure resulted in restoring considerable abductive power to a previously paralyzed lateral rectus.

CASE REPORT

R. M., a 36-year-old Negro, was first seen at the Illinois Eye and Ear Infirmary of the University of Illinois on August 22, 1955. He gave a history of diplopia of one-year duration and stated that his eyes had crossed since that time and that they were gradually becoming more crossed. Corrected visual acuity was 20/30 in the right eye and 20/20 in the left. Examination showed an esotropia of 50 degrees which was slightly greater with the right eye fixing. There was no abduction of the right eye beyond the midline, and the left eye showed a definite weakness of abduction.

The blood and spinal fluid serology were positive. Neurologic examination was negative except for the paralysis of the right lateral rectus and weakness of the left lateral rectus.

There was no change in the extraocular muscle function following an extensive course of penicillin therapy. It was felt that surgery was indicated, and on March 28, 1956, a 10-mm. resection of the right lateral rectus and a four-mm. recession of the right medial rectus were done. The esotropia was improved, but a residual convergence of 25 prism diopters for distance and 30 prism diopters for near remained with no abduction past the midline. On May 10, 1956, a five-mm. recession of the left medial rectus was performed without objective or subjective improvement.

After allowing for postoperative stabilization, a Hummelsheim operation of the right eye was done on November 15, 1956. At operation the previously resected right lateral rectus was isolated and the adhesions from the previous operation were freed with little difficulty. Lateral slips from both the superior and inferior recti were brought to just behind the insertion of the lateral rectus. They were sutured to this site, and the lateral rectus was advanced two mm. without resection. No more was done purposely so that the effect of the transplantation alone could be compared with the effect of the previous resection and recessions.

From the first postoperative day the eyes were straight for distance and near by the cover test. The eyes remained straight and when seen five months after surgery the right eye could be abducted 20 degrees. By cross-cover measurements, there were six prism diopters of esophoria at distance and three prism diopters of esophoria at near. Ten months after surgery the right eye could be abducted 30 degrees.

* From the Department of Ophthalmology of the Illinois Eye and Ear Infirmary, University of Illinois College of Medicine. Presented before the Chicago Ophthalmological Society, November 18, 1957.

Figs. 1, 2 and 3* (Smart and Snydacker). One month before Hummelsheim operation. (Fig. 1) Right lateral gaze. (Fig. 2) Straight-ahead position. (Fig. 3) Left lateral gaze.

* The preoperative photographs were taken by Martin Urist, M.D., and the postoperative photographs were taken by Mr. Louis Pedigo.

Figs. 4, 5, and 6 (Smart and Snydacker). Six months after Hummelsheim operation. (Fig. 4) Right lateral gaze. (Fig. 5) Straight-ahead position. (Fig. 6) Left lateral gaze.

Summary

This case is presented to show that transplantation of one half of the superior rectus and one half of the interior rectus to the insertion of the lateral rectus can be an effective procedure in selected cases.

219 Dallas East Medical Center (18).
111 North Wabash (2).

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EMBOLI OF THE CENTRAL RETINAL ARTERY AFTER MITRAL COMMISSUROTOMY*

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(Translated by David Shoch, M.D., Chicago, Illinois)

An arterial embolus is a relatively frequent complication of mitral stenosis. This accident can occur spontaneously, outside of periods of complete asystole, without emotional upset or effort. It is in the area of the left middle cerebral artery that the embolus is most frequently lodged. Emboli in peripheral arteries are more uncommon and are found, in general, in the legs.

Localization in the central retinal artery is even more rare. Von Graefe described this in 1859 and attributed it to a cardiac lesion.

Mitral commissurotomy is a procedure which consists of stretching the stenosed valve by means of a finger. It is performed on valves which have calcareous concretions on them and which frequently liberate emboli to different areas (H. E. Bolton, et al., 1952). Two of these interest the ophthalmologist: (a) localization in a nutrient vessel of the optic nerves (J. E. Alfano, et al., 1957), and (b) localization in a central retinal artery which to our knowledge has not been described up to now. We should like to report two such cases.

* From the Ophthalmologic Clinic of the University of Liege, Prof. R. Weekers.

Case reports

Case 1

L. Adolphe, aged 31 years, had acute polyarticular rheumatic fever in 1942 without sequelae until 1953 when he had his first attack of pulmonary edema. A diagnosis of mitral stenosis was made and the patient became unable to work because of increasing dyspnea and further attacks of pulmonary edema. In March, 1955, a mitral commissurotomy was performed, the surgeon noting calcifications on the valve.

On awakening the patient noted that he could not see out of his right eye. Vision was reduced to light perception, the fundus showed a pale disc, arteries very narrowed, a pale edema of the posterior pole, a red spot in the macula surrounded by fine hemorrhages and small exudates. A diagnosis of spasm of the right central retinal artery was made. The patient left the hospital on April 7th and was seen on the eye service on May 9th. The right eye was blind, the disc atrophic, and the narrowed arterioles converted to white cords. There were scattered areas of degeneration in the papillomacular area and the macula, still red, seemed to show a hole.

CASE 2

B. Alexandre, aged 36 years, had acute rheumatic fever in prison camp in 1943 at the age of 22 years. Two years later he presented cardiac complications attributable to a mitral stenosis which prevented him from working. In December, 1957, a mitral commissurotomy was performed. The surgeon did not find any clots in the auricle but did note calcifications on the valve. On awakening the patient noted a diminution of vision in his left eye. Vision was reduced to hand movements in the temporal paracentral area. The pupil was semidilated, reacted feebly to direct light but strongly to a light in the contralateral eye. The retina showed a papillomacular edema, arterioles in spasm, and the veins irregular. A cherry-red spot was seen at the macula. The disc was pale, its borders indistinguishable in the surrounding edema.

The patient was seen by the eye service on December 30, 1957, and the function and ophthalmoscopic appearance of the left eye remained unchanged.

On January 27, 1958, the foveal reflex had disappeared and the disc had become pale. Visual fields showed an absolute central scotoma. Vision remained limited to hand movements, the left pupil was larger than the right, and reacted feebly to light.

On March 3, 1958, the condition remained unchanged. The disc was grossly atrophic, vessels narrowed, and the macular zone poorly differentiated.

These two cases occurred in a series of 46 mitral commissurotomies performed on the surgical service of Hôpital de Bavière in Liege (Prof. L. Christophe and F. Orban).

SUMMARY

A description of two cases of embolism of the central retinal artery occurring after mitral commissurotomy performed for severe mitral stenosis.

66 Boulevard de la Constitution.

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CHLOROMA OF THE ORBIT

A REPORT OF FOUR CASES

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Chloroma is a rare type of leukemic manifestation in children. In its most characteristic form it is associated with greenish-colored tumors which are usually subperiosteal and are found specially in relation to the bones of skull and thorax. In the cases to be described, the first one showed more or less every clinical feature of the disease, while the other three cases did not because of unilateral orbital involvement. This could also have developed to the other side if the patients had survived for a longer time. The interesting feature about the second case was proptosis of the right side, with enlargement of the submandibular lymph nodes and parotid gland and facial paralysis of the left side. Except in the first case, the blood, bone marrow, and X-ray findings appeared some time after the proptosis. Involvement of the parotid gland and facial paralysis are not common features of the disease.

CASE REPORTS

CASE 1

J. B., a one-year-old boy, came to the hospital with a history of fever for a few days, a month earlier, after which a rapidly increasing prominence of the eyes was noted. A fullness over the temple had also been noted for the last three days. The child could not close his eyes because of the proptosis. There was discharge from the nose and the child was extremely irritable.

On examination (fig. 1), he showed typical chloroma findings—the temples were full and there was bilateral proptosis, more marked on the left side. The maxillary prominences were more marked and the palate was depressed and showed a greenish growth. The nares were blocked. The temporal swellings were of a dirty green color over which veins were prominent. The swellings were adherent to the deeper structures. Submandibular and neck glands were enlarged and hard. There was a palpable enlargement of the liver and spleen.

Local examination revealed normal orbital mar-

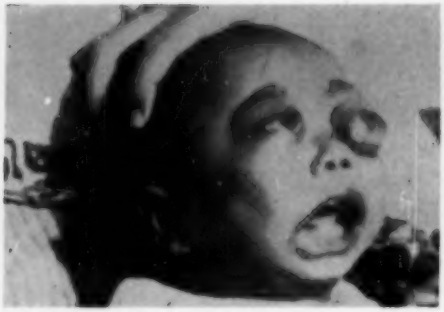

Fig. 1 (Mathur). *Case 1.* Findings typical of chloroma.

gins. Retrobulbar swellings were greenish, rubbery hard, and connected to the orbits. Eyelids were swollen. Chemosis and exposure keratitis were marked in the left eye. The condition of the eye was normal except for some restriction of movements. Fundi did not reveal any changes.

The patient developed a temperature up to 101°F. three days after the examination, which came down to normal with penicillin.

Laboratory investigations showed: Blood: E.S.R., 23 mm. after one hour (Westergren); hemoglobin, 6.5 gm. percent; R.B.C., 29,20,000 per cmm.; W.B.C., 14,000 per cmm.; blast cells, two percent; myelocytes, 11 percent; polymorphs, 26 percent;

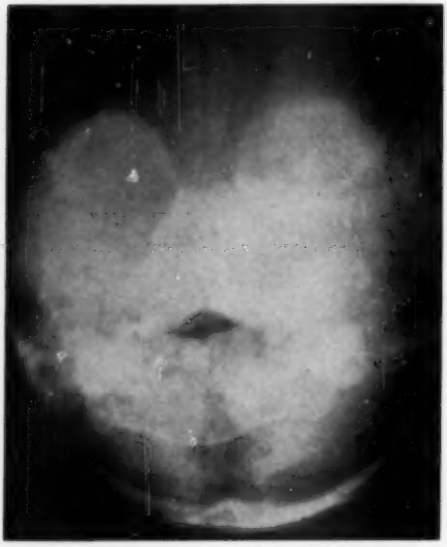

Fig. 2 (Mathur). *Case 1.* X-ray films showed a soft tissue shadow involving the bones of face, orbits, and base of the skull.

Fig. 3 (Mathur). *Case 1.* Another X-ray view.

lymphocytes, 60 percent; large monocytes, one percent.

X-ray films showed chest and long bones to be normal; skull, a soft tissue shadow involving the bones of face, orbits, and base of the skull (figs. 2 and 3). Bone narrow smear: Neutrophils, five percent; eosinophils, one percent; metamyelocytes, eight percent; myelocytes, 21 percent; promyelocytes, eight percent; myeloblasts, nine percent; lymphocytes, 29 percent; monocytes, two percent; others, nil. Erythrocytes; normoblasts, 15 percent; pronormoblasts, two percent.

Biopsy. The tissue was dirty green in color with a few punctate hemorrhages over the surface. The microsection (fig. 4) showed three or four irregular small bony trabeculae and the rest was made up of a dense sheet of cells. They did not occur in any follicular arrangement and had displaced all soft and bony tissue. Under high power these cells were seen to contain large hyperchromatic nuclei which had almost completely replaced the cytoplasm. The cytoplasm was scanty, mostly basophilic in the paraffin section. The cell type could not be distinguished, but the fact of immaturity of nuclei and densely packed cells which displaced all the bony tissue pointed to a leukemic nature.

CASE 2

S., a four-year-old girl, came to the hospital with a history of injury about a week before. Slight proptosis of the right eye appeared gradually. The skin of the lower lid showed a dirty green discoloration. Local and general examinations did not reveal anything except for some irritability and the pale look of the child. The patient was admitted to the hospital for observation.

After 10 days the proptosis was marked with a

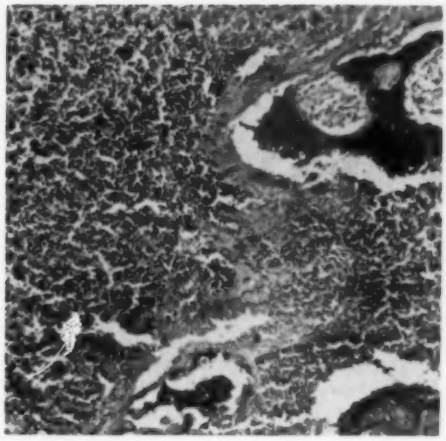

Fig. 4 (Mathur). *Case 1.* Photomicrograph of biopsy.

bluish-green discoloration of the lower lid. Orbital margins were normal and the retrobulbar swelling was palpable. Upper cervical and submandibular lymph glands of the left side were enlarged, as was the parotid salivary gland which was hard and discrete. There was left-sided facial paralysis. The palate showed a greenish discoloration on the right side, and the right nostril was obliterated. There was fullness of the right temple.

After another five days, the right fundus showed engorged tortuous veins, marked retinal edema, two vacuolated and one massive flame-shaped hemorrhages. The left eye developed a small subconjunctival hemorrhage, while the fundus showed some venous engorgement.

Later proptosis increased and the growth extended below the lower orbital margin onto the face, with prominent veins in the overlying skin. The fullness of the temple increased and the palate was depressed on the right side. The temperature rose to 102°F. but was controlled with penicillin.

Laboratory investigations. Blood: E.S.R., 28 mm. after one hour (Westergren). Hemoglobin, 5.5 gm. percent; R.B.C., 22,50,000 per cmm.; W.B.C., 19,400 per cmm.; blast cells, nil; myelocytes, six percent; polymorphs, 25 percent; lymphocytes, 68 percent; large monocytes, one percent.

X-ray studies. Chest, long bones, and skull were normal. Sternal puncture smear: myeloblasts, two percent; lymphocytes, 20 percent; lymphoblasts, 70 percent; polymorphs, nil; large monocytes, nil; eosinophils, two percent; normoblasts, three percent, other immature cells, three percent.

Biopsy showed a picture identical to the one described in Case 1.

Case 3

S., aged seven years, a boy, was admitted to the hospital with proptosis of left eye and fever of seven days' duration.

General examination revealed a palpable enlargements of liver and spleen; cervical glands of both the sides were enlarged; there were facial paralysis of the left side and marked anemia. Temperature ranged from 101°F. to 102°F. The palate was depressed on the left side; the left nostril was blocked; the swelling extended to the left temple.

Local examination of the right eye showed nothing abnormal. The left eye was proptosed, with marked chemosis, an exposed hazy cornea, movements very much restricted, and a palpable growth in the retrobulbar tissues. The fundus of the left eye could not be seen. That of the right eye was normal.

Laboratory investigations. Blood: E.S.R., 102 mm. after one hour (Westergren). R.B.C., 17,50,000 per cmm.; W.B.C., 10,600 per cmm.; immature cells, 63 percent; polymorphs, two percent; lymphocytes, 35 percent. X-ray films: Chest and long bones, normal; a soft tissue shadow was visible over the facial and orbital bones. Bone marrow smear; myelocytes, three percent; metamyelocytes, three percent; normoblasts, three percent; lymphocytes, 11 percent; lymphoblasts, 71 percent; myeloblasts, seven percent; other immature cells, two percent.

Biopsy. Both gross and microscopic pictures were typical of chloroma.

Case 4

G., a three-year-old girl, was admitted with right-sided proptosis for the last 15 days and fever for six days. General examination revealed enlargement of cervical glands and facial paralysis of the right side, slight anemia, bluish coloration of the palate but no depression, and blocked nares. The left eye was normal, while the right eye showed marked proptosis, chemosis, exposure keratitis, movements very much restricted, and a retrobulbar growth which could be palpated. Fundi of both the eyes showed flame-shaped hemorrhages of various sizes, with engorgement of veins.

Laboratory investigations: Blood: E.S.R., 98 mm. after one hour (Westergren). R.B.C., 20,25,000 per cmm.; W.B.C., 15,500 per cmm.; immature cells, 52 percent; polymorphs, 30 percent; lymphocytes, 18 percent. X-ray: Chest and long bones, normal; soft tissue shadow was visible over facial and orbital bones. Bone narrow smear: Neutrophils, eight percent; eosinophils, two percent; metamyelocytes, 10 percent; myelocytes, 20 percent; myeloblasts, 12 percent; lymphocytes, 30 percent; normoblasts, 18 percent.

Biopsy. Both gross and microscopic appearance was typical of chloroma.

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OPHTHALMIC RESEARCH

EDITED BY FRANK W. NEWELL, M.D.

Abstracts of papers presented at the meeting of the Eastern Section of the Association for Research in Ophthalmology, Inc., November 21 and 22, 1958, at New York University-Bellevue Medical Center.

Section Chairman: Jerry H. Jacobson, M.D., New York

Experimental radiation cataracts induced by microwave radiation. Russell L. Carpenter, Ph.D., David K. Biddle, B.S., Claire Van Ummersen, B.S., Cayetano P. Mangahas, M.D., and Hal M. Freeman, M.D., Department of Biology, Tufts University, Medford, Massachusetts. This research was supported in whole by the United States Air Force under Contract No. AF 41(657)-86, monitored by the Rome Air Development Center, Griffiss Air Force Base, New York.

To study the conditions under which lens opacities are induced by microwave radiation, eyes of 56 male New Zealand white rabbits were exposed to continuous-wave radiation at a frequency of 2450 mc. and wave lengths of 12.3 cm. for various periods of time and at power densities ranging from 0.12 watts/cm.^{2} to 0.40 watts/cm.^{2} Onset and progress of the resulting posterior subcapsular cataracts were observed by ophthalmoscope and slitlamp and thresholds for opacity formation established in terms of time and power densities. It was apparent that the higher the power, the shorter the single exposure period required to induce opacities. In initial appearance and in morphology, cataracts were similar to those described by Cogan and Donaldson (Arch. Ophth., **45**:1, 1951) as resulting from ionizing radiation but the latent period following microwave radiation was much shorter, averaging three and one-half days.

Temperatures of the vitreous body during irradiation at five different power levels were measured by a thermistor probe positioned directly behind the posterior pole of the lens. At higher power densities, vitreous temperatures increased more rapidly than at lower powers and reached a higher level before tending to level off; that is, the more energy applied per unit of time, the greater and more rapid the thermal effect from absorption of that energy. Relating vitreous temperatures to time and power thresholds, it appeared that at each power level the threshold time for opacity formation coincided with the time of exposure at which the vitreous temperature approximated 50°C.

Having established that at 0.28 watts/cm.^{2} the minimum single exposure period which was cataractogenic was six minutes, cumulative effects of repeated subthreshold exposures at this power were studied. Irradiations of five, four and three minutes' duration were repeated at intervals of one day, two days, one week or two weeks. Opacities resulted from as few as two to five exposures in 34 of 43 experiments. Two four-minute exposures two weeks apart proved cataractogenic. In 14 cases, partial or complete regression of the opacity occurred; in the remainder, it persisted unchanged for periods up to a year.

To ascertain whether or not the effect of microwave radiation on the lens is purely thermal, the same microwave source was employed to generate pulsed wave instead of continuous wave radiation. On a 0.50 duty cycle, whether the pulse repetition rate is 200, 1,000 or 5,000 per second, the eye is subjected to microwave radiation for only half the time during any given exposure period. Thermal effects were found to be related to average power density. Thus, with radiation pulsed on a 0.50 duty cycle at a peak power of 0.28 watts/cm.^{2}, the average power density was 0.14 watts/cm.^{2} and the temperature of the vitreous body increased at the same rate and to the same extent as when the eye was subjected to continuous wave radiation at 0.14 watts/cm.^{2} Under these conditions, a 20-minute exposure was equivalent in terms of microwave energy to a 10-minute exposure to continuous wave radiation at 0.28 watts/cm.^{2} power density, which had been shown to be cataractogenic. In terms of thermal effect, it was identical to a 20-minute exposure to continuous wave radiation at 0.14 watts/cm.^{2}, which had been proven insufficient to cause opacity formation. Twelve animals were exposed to pulsed wave radiation under the conditions described above and lens opacities developed in eight of them. Control animals exposed for 20 minutes to continuous wave radiation at 0.14 watts/cm.^{2} power density did not develop opacities. This preliminary series suggests the probability that the cataractogenic effect of microwave radiation at this wave length is not primarily thermal.

Serial antistreptolysin-O determinations in patients with uveitis. Robert S. Coles, M.D., and Arthur Nathaniel, M.D., New York University Post-Graduate Medical School, New York.

While anterior, or nongranulomatous, uveitis has long been considered to represent an allergic reaction of the delayed bacterial or tuberculin variety, only recently has the streptococcus been implicated as the prime etiologic antigenic agent. This evidence is based, in part, on the finding of higher antistreptolysin-O (ASLO) titers in patients with nongranulomatous uveitis than in those with granulomatous uveitis or in control subjects.

This finding, however, has not been generally confirmed. In this country there has been only one published report on antistreptolysin-O titers and uveitis, and several contradictory reports have appeared in the European literature. This study was undertaken to determine if serial antistreptolysin-O measurements on patients with uveitis and in a control group would more clearly define the association of nongranulomatous uveitis and the streptococcus.

For this study a total of 301 antistreptolysin-O determinations were performed on 68 patients with uveitis. Of these, 116 determinations were in 36 patients with anterior uveitis, and 89 in 32 patients with posterior uveitis. Only random antistreptolysin-O titers were determined on 96 control subjects. For the purpose of this study we accepted a titer of 125 units as the upper limit of normal.

Our results indicate that there is no appreciable difference in the antistreptolysin-O titers in the three groups. In the patients with anterior uveitis, serial testing revealed that 86 percent had titers of 125 units or less; in patients with posterior uveitis, 85 percent; and in the control group, 89 percent. In only five patients in this study was there a suggestive correlation between the severity and acuteness of the disease and an elevated titer which later reverted to normal during the course of serial testing. However, four of these were in patients with posterior uveitis and only one was a patient with anterior uveitis. This is at variance with previous reports that elevated antistreptolysin-O titers are more commonly found in anterior uveitis.

Our data do not support the thesis that there is an association between antistreptolysin-O titers and anterior or posterior uveitis. By inference, we feel that there is no causal relationship between streptococcal infection and anterior uveitis.

Spectral sensitivity (distribution) of color-defective individuals determined by electroretinography. R. M. Copenhaver, M.D., R. D. Gunkel, O.D., and P. Gouras, M.D., Ophthalmology Branch, National Institute of Neurological Diseases and Blindness, National Institutes of Health, U.S. Department of Health, Education and Welfare, Bethesda, Maryland.

The eyes of color-defective volunteers were stimulated with flickering monochromatic light (32 per second) obtained by interposing a rotating sector disc and double interference filters of 15 different wavelengths before a xenon arc light. The photopic electrical potentials of the retina were recorded with a direct-reading encephalograph. Light stimuli flickering at a rate faster than the scotopic activity can follow were used so that only photopic potentials were recorded. From the magnitude of the electroretinal responses obtained at different wave lengths the relative spectral sensitivities of the photopic component were calculated.

Dr. Eberhard Dodt initiated the present study at the National Institutes of Health with an investigation of two protanopes and two protanomalous subjects, which has been reported. The totally red-blind individuals showed a shift in maximum sensitivity from 558 mμ to 531 mμ with a pronounced reduction in sensitivity on the long wave length side of the spectrum, while the partially red-blind individuals showed a decrease in red sensitivity which was intermediate between that of the normal and the protanope. With the use of large stimulus areas (60°) instead of small (1°) an increased blue sensitivity was found with a maximum at 460 mμ which is thought to be due to a blue-sensitive photo-pigment and not visual purple absorption.

Five additional protanopes have been tested, and the findings confirm the marked loss in red sensitivity and the shift of the sensitivity maximum to a slightly shorter wave length. A greater blue sensitivity was found in the protanope than in the normal, particularly when a larger area of retina was stimulated.

Five deuteranopes were examined and showed a shift of maximum sensitivity from 558 mμ to 583 mμ and a distinct reduction in green sensitivity. The findings on several deuteranomalous patients and several subjects with large central chorioretinal lesions will also be shown. The various findings will be discussed.

The active transport of sodium across the rabbit corneal epithelium. Anthony Donn, M.D., and Nancy L. Mills, B.S., Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York.

A modification of Ussing's technique was employed to measure simultaneously the influx and outflux of sodium ions across the rabbit corneal epithelium. An excised rabbit cornea, clamped between two lucite blocks and bathed with a physiologic solution, generated an electrical potential up to 40 millivolts. While this potential difference was short-circuited with a known current the influx and outflux of sodium ions across the epithelium were measured simultaneously, using Na^{24} and Na^{22}.

It is demonstrated that there is active transport of sodium ions across the rabbit corneal epithelium. Sodium is "pumped" from the tear fluid side into the aqueous humor side of the corneal epithelium.

The ocular penetration and tolerance of a new antibiotic: Spiramycin. Francis P. Furgiuele, M.D., Theodore W. Sery, Ph.D., and Irving H. Leopold, M.D., Wills Eye Hospital, Philadelphia. (Ciba Pharmaceutical Products Inc. made available the Spiramycin used in this study.)

A new antibiotic, Spiramycin, derived from Streptomyces ambofaciens was tested in albino rabbit eyes for tolerance and penetrability. The tube serial dilution method was employed using

B. subtilis A.T.C. #6633 as the test organism.

Toxicity and tolerance. On normal eyes, topical application of solutions of Spiramycin (0.5-5.0 percent) and ointments (1.0 to 5.0 percent) were well tolerated. Instillation of a five-percent solution or ointment on eyes whose epithelial barrier was damaged, exhibited mild conjunctival hyperemia, chemosis and exudation which was transient and reverting to normal within 24 to 36 hours. More marked and persistent tissue reactions as mentioned above occurred with 10-percent solutions of the drug when applied to both normal and abraded corneas.

The powdered Spiramycin dusted on the normal eye was moderately toxic (chemosis, hyperemia, clouding of epithelium) after two to three hours with reparation within 48 hours. On eyes with partially de-epithelialized corneas pure powder Spiramycin produced marked local damage to the cornea and conjunctiva.

Intracameral and subconjunctival injections. These were well tolerated and nontoxic in amounts up to 5,000 µg. for the subconjunctival route, and 250 µg. injected into the anterior chamber.

Intravitreal injections. Direct injection of buffered solutions into the vitreous in 100, 250 and 500 µg. amounts were well tolerated with no notable toxic effects. Higher quantities between 1000 and 2500 µg. produced exudative reaction within the posterior segment of the eye with clouding of posterior lens capsule.

Penetration. In the presence of a normal eye topical application of solutions (one to five percent) and ointments (one to five percent) gave no detectable penetration of Spiramycin in the aqueous; 10-percent solution topically showed slight aqueous penetration after one hour with no detectable amounts after two hours. Subconjunctival injections of 10,000 µg. gave sustained levels in the aqueous one, two and four hours after injection; 5,000 µg. via this route however showed no detectable drug in the aqueous.

On eyes with abraded corneas drop application of a four-percent solution given as three drops every five minutes for one-half hour produced 64 µg. of Spiramycin per cc. of aqueous detectable after one hour.

A five-percent ointment preparation also showed satisfactory penetration in the aqueous when applied on the abraded cornea.

Intravenous dosages ranging from 10,000 to 200,000 µg. per injection did not penetrate the aqueous or vitreous of the normal eye although significant levels were recoverable in the secondary aqueous.

When either the anterior or posterior segment alone was artificially inflamed, aqueous, but not vitreous, penetration of the drug did occur following large intravenous doses.

Therapy. The high degree of sensitivity of B. subtilis to Spiramycin suggested it might be of value in treatment of vitreous abscess due to this organism. To date, therapeutic trial of intravitreous injections of Spiramycin into experimentally induced vitreous infections with B. subtilis suggested improvement.

Biochemical studies on the mitochondria of retina. Sidney Futterman, Ph.D., and Jin H. Kinoshita, Ph.D., Massachusetts Eye and Ear Infirmary, Boston.

Retinal mitochondria were prepared from homogenates of cattle retina in 0.25 M sucrose containing 3.5×10^{-3} M ethylenediaminetetraacetic acid and fortified with adenylic acid, phosphate, magnesium, and cytochrome-C oxidized tricarboxylic acid substrates. When ethylenediaminetetraacetic acid was omitted from the medium the mitochondria showed a decreased capacity to oxidize all substrates with the exception of succinate. DPNH and TPNH cytochrome-C reductase activities, and cytochrome oxidase were demonstrated.

Electroretinography in night-blinding disorders. George Goodman, M.D., Manhattan Eye, Ear and Throat Hospital, New York, and Ralph D. Gunkel, O.D., Ophthalmology Branch, National Institute of Neurological Diseases and Blindness, Bethesda, Maryland.

Electroretinography was studied in Oguchi's disease, congenital night blindness, and other retinal night-blinding disorders. The scotopic electroretinographic responses was compared to estimates of scotopic function obtained from dark adaptometry and perimetric light sense testing. Patients with similar degrees of scotopic deficit on psychophysical testing showed a wide variation in the amplitude of their scotopic electroretinographic response. The physiologic implications of these findings are considered, and the role of the electroretinogram in the differential diagnosis of night-blinding diseases is discussed.

Studies on the mechanism of action of Diamox. Harry Green, Ph.D., and John L. Sawyer, Wills Eye Hospital, Philadelphia.

The dynamics of exchange of the bicarbonate ion between the arterial plasma and aqueous humors of anterior and posterior chambers of the rabbit eye were studied with the use of intravenously injected C-14 labeled $NaHCO_3$. The results with normal, untreated animals indicated that the bulk of the bicarbonate ion in the aqueous humors was derived directly from the plasma and that the ion was transferred as such into the aqueous humors. The contribution of the $CO_2 + H_2O \rightleftharpoons H_2CO_3$; $H_2CO_3 \leftrightarrow H_+ + HCO_3$. is considered to play only a minor role in the accumulation of bicarbonate ion in the aqueous humors. The effect of Diamox upon the dynamics of the bicarbonate ion was also studied with the use of C-14 labeled $NaHCO_3$. The results showed that the drug suppressed the transfer of bicarbonate ion into the aqueous humors within five minutes after injection. While this rapid action of Diamox upon the transfer of bicarbonate ion into the

aqueous humor correlates well with its equally rapid hypotensive action, described by Gloster and Perkins, it does not correlate well with the time sequence for the inhibition of carbonic anhydrase in the anterior uvea. The results tend to question the validity of the carbonic anhydrase theory of the formation of aqueous humor and the mechanism of action of Diamox in this regard.

Osmotic pressure measurements of intraocular fluids by an improved cryoscopic method: Physiologic significance relative to aqueous humor dynamics. Miryam Z. Kass, Ph.D., and Harry Green, Ph.D., Wills Eye Hospital, Philadelphia.

The freezing point apparatus designed by Ramsey and Brown (J. Sc. Instruments, 32:372, 1955) was constructed with several minor modifications designed to increase the sensitivity, reproducibility and accuracy of the instrument. This cryoscopic assembly permitted the determination of volumes of the order of 10^{-3} µl. and was arranged so that samples of blood plasma, and aqueous humors of the anterior and posterior chambers from the same animal could be studied simultaneously in the same system under exactly the same bath and temperature conditions. The freezing point depressions of the individual fluid samples were used as estimates for the determinations of their relative osmotic pressures.

The results with more than 20 animals suggest the absence of a significant osmotic pressure gradient between the plasma and the aqueous humor of the posterior chamber. It is quite clear, however, that the osmotic pressure of the aqueous humor of the anterior chamber is not only definitely higher than that of the plasma, but also significantly higher than that of the posterior chamber fluid.

The effect of Diamox upon the osmotic pressures of the three fluids was also studied. The intravenous injection of 30 mg./kg. of Diamox was found to have no apparent effect upon the osmotic pressure of any of the three fluids studied after 30 minutes.

The results of this investigation indicate that while some doubt may exist as to the existence of an osmotic pressure differential between the posterior chamber aqueous humor and the plasma the evidence strongly indicates the existence of a higher osmotic pressure in the aqueous humor of the anterior chamber. The implications of these results relative to the mechanism of formation of aqueous humor and the action of Diamox thereon will be discussed.

Pharmacology of Daraprim. Herbert E. Kaufman, M.D., and Lee A. Caldwell, Phar.B., and Jack S. Remington, M.D., Ophthalmology Branch, National Institute of Neurological Diseases and Blindness, and the Laboratory of Tropical Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland.

The pharmacology of pyrimethamine (Daraprim, Burroughs-Wellcome) was studied in 19 patients by means of serial serum bioassays. Great differences were observed in the serum concentration of pyrimethamine in patients taking identical amounts of the drug. These differences were independent of the weight of the patient. Since the rate of disappearance of the drug from the serum upon cessation of therapy was similar for all patients and the rates of excretion and metabolism of the drug were similar, the differences in serum concentrations observed were presumably due to variations in intestinal absorption.

These studies indicate that pyrimethamine is slowly destroyed and that a large "loading" dose and a smaller "maintenance" dose provide most stable blood levels. The administration of Daraprim, 100 mg. twice the first day and 25 mg. twice a day thereafter, provides sustained high blood levels with minimal toxicity. Patients treated in this way achieve stable blood levels of the drug approximately two weeks earlier than when 25 mg. twice a day is given from the outset.

In those patients developing hematologic toxicity, the serum level of Daraprim was significantly higher than in unaffected patients.

The effect of pontocaine on the carbohydrate metabolism of the bovine corneal epithelium. Jin H. Kinoshita, Ph.D., Harvard Medical School and Howe Laboratory, Boston, Massachusetts.

Herrmann and Friedenwald have previously shown that pontocaine inhibits the respiration and glucose metabolism of the cornea. Probably due to this interference in the energy metabolism of the cornea, this anesthetic exerts an inhibitory effect on the regeneration of the epithelium. An attempt was made, therefore, to establish the stage in the glucose metabolism where the inhibition may occur. In this study, although we were unable to establish the exact site of action of pontocaine, considerable information regarding the details of carbohydrate metabolism of the bovine corneal epithelium was gained.

For these studies, variously labeled C^{14} glucose and pyruvate were used as the substrate. The oxidation of each of these substrates to carbon dioxide by the isolated bovine corneal epithelium was effectively inhibited by the proper concentration of pontocaine. From these experiments it was learned that pontocaine blocked the two aerobic pathways, the citric acid cycle and the hexose monophosphate shunt pathway, but did not affect the anaerobic process, the Embden-Meyerhof scheme.

To study further the stages of the carbohydrate metabolism, homogenates and mitochondria preparations of the corneal epithelium were employed. By this technique it was possible to follow experimentally the transfer of electrons from the substrate to the pyridine nucleotides, to cytochrome-C, to cytochrome oxidase and then to oxygen. Pontocaine did not seem to inhibit at any of these oxidative stages. The possible ex-

planations for failing to detect the exact site of inhibitory action of pontocaine will be discussed.

The reactivity of sulfhydryl groups in bovine lenses. Jin H. Kinoshita, Ph.D., and Lorenzo O. Merola, Harvard Medical School and Howe Laboratory, Boston, Massachusetts.

It has been shown that in experimentally produced cataracts there is a marked decrease in the -SH level. The disappearance of the -SH groups of lens protein has been attributed to an oxidative process resulting in the formation of disulfide linkages. Because of the difficulty of assaying the protein -SH groups, very little information is available about their reactivity and factors which influence their level in the normal lens.

The reactivity of the -SH groups in lens homogenates was studied by the amperometric titration procedure and by the rate of oxidation of these groups. The glutathione in the lens homogenates seems to be much more reactive than the protein -SH groups. Apparently there exist varying degrees of reactivity of protein -SH groups. There are those which are only observable when measured in the presence of 8.0 M urea. The age of the lens also influences the state of reactivity of these groups. In the mature cattle lens almost all of the protein -SH groups are masked in some manner while in the younger calf lens they appear much more reactive.

Pathogenesis of senile macular degeneration. A. L. Kornzweig, J. Schneider, and M. Feldstein, New York University-Post Graduate Medical School, M. Sinai Hospital, and Home for Aged and Infirm Hebrews, New York.

A pathologic examination of 65 eyes of elderly residents of the Home for Aged and Infirm Hebrews of New York City was made. All these patients had senile macular degeneration of varying degrees of severity. Three different grades of severity were noted and demonstrated by photomicrographs of serial sections.

The pathologic changes demonstrated show a gradual but persistent progression of an atrophic process affecting the cone cells and the association fiber cells in the region of the fovea centralis and surrounding macular area. A reactive process in the adjacent pigment layer results in loss of pigment granules, proliferation and migration of pigment cells. Cystic spaces in the fovea and nerve fiber layer also indicate areas of atrophy of cell substance and, possibly, also areas of exudation, the contents having been dissolved out or lost in the process of fixation, embedding and staining of the sections.

The hypothesis is presented that this area of the retina acts like a peripheral vascular end-organ. Its gradual deterioration is believed to be due to a slow obliteration of the blood supply and consequent anoxia and loss of nutrition, partly from the adjacent choriocapillaris and partly from the retinal circulation. The retinal circulation may be of greater importance in the condition than it was thought to be heretofore.

Demecarium bromide (BC-48): A new anticholinesterase agent in treatment of glaucoma. Narenda Krishna, M.D., Wills Eye Hospital, Philadelphia.

Anticholinesterase agents are known to lower the intraocular pressure. Demecarium bromide (BC-48), a new anticholinesterase agent, has been investigated for its therapeutic value in glaucoma. Preliminary to such an investigation, its effect on rabbit eyes and normal human eyes was studied.

In rabbit eyes, BC-48 produces intense miosis, lowering of intraocular pressure after an initial transient rise and increase in the facility of aqueous outflow when instilled into the rabbit eyes. A one-half percent concentration produces conjunctival congestion and has been found to be lethal to rabbits. The local application does not cause permanent damage to the eyes of rabbits.

Encouraged by the preliminary studies in rabbit eyes BC-48 has been studied for its effects on normal human eyes. Local instillation in normal human eyes produces intense miosis, accompanied by browache, blurred vision and pain, with significant lowering of intraocular pressure and increase in aqueous outflow, which last for several days.

Nearly 100 glaucomatous eyes of various types of glaucoma have been treated with 0.1 and 0.25-percent BC-48 for a period of several days to several months. Repeated tension measurements, tonographic studies, visual fields and gonioscopic examinations have been taken for control. Based on the analysis of these cases, BC-48 in the strength of 0.1 percent and 0.25 percent from one instillation every other day to twice a day promises to be a useful therapeutic agent for the treatment of glaucoma.

Histochemical study of dehydrogenase activities of the retina. Toichiro Kuwabara, M.D., and David G. Cogan, M.D., Howe Laboratory of Ophthalmology, Boston.

The reduction of neotetrazolium by intact human, rabbit, and cattle retinas was observed after aerobic and anaerobic incubation over a wide pH range in the presence of various substrates and cofactors. Two general patterns of tetrazolium reduction could be obtained in frozen sections of the retina prepared after incubation.

One pattern, observed with tricarboxylic acid substrates, and most prominently demonstrated when succinic acid was the substrate, consisted of a scattered precipitate of blue tetrazolium in the nerve fiber layer and in the reticular layer, with considerable amounts in the ellipsoids of the photoreceptors.

A second pattern of insoluble formazan was obtained with DPNH or lactate as substrates, and in the latter case required the addition of DPN.

The dye was concentrated in Müller's cells forming a heavy plexus of stained fibers just beneath the internal limiting membrane with fibers extending toward the outer layers of the retina. No dye reduction occurred in the photoreceptors. There was considerable species variation and this pattern was most prominently seen in human retinas, particularly in eyes with absolute glaucoma.

These observations suggest that Müller's cells and fibers, heretofore thought to have structural functions only, may serve a significant metabolic role.

Ocular effects of endotoxin. R. Z. Levene, M.D., and G. M. Breinin, M.D., New York University Post-Graduate Medical School, New York.

The effects of endotoxin on the normal rabbit eye have been investigated. Endotoxin in small amounts can produce an ocular inflammation with alterations in the intraocular pressure and aqueous flow rate. Histologic study demonstrates edema and hemorrhages of the ciliary processes and generalized leukocytic infiltration. Pretreatment with cortisone completely inhibits the in vivo ocular effects although pretreatment with compound 48/80 has no protective effect. Tolerance to the ocular inflammatory effect can be quickly developed. The ubiquity of endotoxin as a contaminant and the importance of recognizing its ocular effects in the normal eye are stressed.

Pupillary reactions in normal man: In response to light stimuli of threshold intensity. Otto Lowenstein, M.D., and Irene E. Lowenfeld, Ph.D., Columbia University, College of Physicians and Surgeons, New York.

The movements of both pupils of normal subjects were continuously recorded with the electronic pupillograph.

After 10 to 30 minutes of dark adaptation, one eye was exposed to intermittent light stimuli of fixed area, threshold intensity and variable duration and frequency. The visual and the pupillary thresholds were compared in each case. Reflex characteristics such as latency period, speed, extent and duration of contraction and redilatation were compared with those of pupillary reflexes to brighter light stimuli.

The findings, which differ from those of recently published reports, will be discussed in terms of their theoretical implications and in relation to possible application of such methods for the detection of pathology within the retina, optic nerve, chiasm and tract.

Small, cytoplasmic elements in lens fibers: Integrated biochemical and electron microscopic observations. Robert A. Resnik, Ph.D., Theodor Wanko, M.D., Mary Ann Gavin, M.S., and Edith B. Kenton, B.S. Ophthalmology Branch, National Institute of Neurological Diseases and Blindness, Bethesda, Maryland.

In this study the cytoplasmic constituents of the fibers of rabbit and calf lenses were separated by differential centrifugation. Fixed and embedded samples of the following fractions were examined with the electron microscope: mitochondrial, microsomal, first and second postmicrosomal residues and the supernant (P-5-S) from the second postmicrosomal fraction.

Chemical investigation was primarily concerned with P-5-S since morphologic examination of this fraction disclosed an element of filamentous appearance only, which is the most abundant structure in the cytoplasm of the lens fiber. Its characteristics, in P-5-S, of low density to electrons, indefinite length, and a diameter of about 100 Å coincide with those of the filamentous structures seen in sections of intact lens fibers.

In an attempt to identify these elements, it was found that they are associated with the protein component of the lens fibers. The chemical characteristics of these elements will be discussed.

Lens proteins II: The effect of pH on alpha crystallin. R. A. Resnik, Ph.D., E. Kenton, B.S., and S. Lourie, Ophthalmology Branch, National Institute of Neurological Diseases and Blindness, Bethesda, Maryland.

Studies on alpha crystallin have not provided any evidence for the existence of the forms of alpha crystallin (alpha$_1$, alpha$_2$, alpha$_c$, and alpha$_a$) proposed by François, et al. A marked pH dependence of alpha crystallin has, however, been demonstrated.

Below pH 4.5, solutions of alpha crystallin undergo a four-fold increase in the intrinsic viscosity. The protein splits into two parts below pH 4. At pH 4.5, 6.0, and 7.0 the sedimentation pattern of alpha crystallin is asymmetric. This asymmetry, which disappears at about pH 7.8, may be due to an aggregation of the protein molecules. It does not appear to be due to the presence of another "type" of alpha crystallin. Different methods of preparing this material all yield protein which has this same characteristic sedimentation pattern.

Above pH 8.3 alpha crystallin in unstable. The presence of small amounts of beta and gamma crystallin do not protect the protein from the changes induced above pH 8.3.

Random occurrence of herpetic disciform keratitis in rabbits. Theodore W. Sery, Ph. D., Wills Eye Hospital, Philadelphia.

The increasing occurrence of disciform keratitis in humans, as a result of complications following a primary or recurrent herpes simplex keratitis, has made it necessary to find a better experimental approach to the study of the disease. The literature contains only isolated reports of the condition as found in experimental animals but no definitive data has as yet appeared which would offer some clues as to the mechanism of its production.

The present paper concerns the observation of 22 cases of the disease as it occurred in a random fashion from a total of 288 corneas that were inoculated with herpes simplex virus. All primary infections were of the typical dendritic ulcer type and usually occurred about the second day after the virus was applied to the abraded epithelium. The majority of these infections were self-limited. Four strains of virus were used; these included the well known HF strain and three others isolated from patients. The disciform opacity in rabbit corneas started to develop as early as seven days and as late as 14 days after inoculation but the greatest number developed between nine and 11 days postinoculation. A tendency was noted, for the cornea that was developing a disciform keratitis, to display four characteristics; these were: (1) opacity of the stroma, (2) thickening of the cornea, (3) vascularization of the cornea, and (4) corneal anesthesia. These characteristics are seen in human cases except that vascularization is less frequently seen in humans and usually occurs very late. In the rabbit, corneal vascularization was a prominent feature and usually preceded by a day or two the start of opacification of the stroma. An exudative reaction in the rabbit was seen in about half of the cases and consisted of a polymorphonuclear reaction but with variable numbers of mononuclear leukocytes. The opaque phase began as an edema that involves half or more of the total stromal depth. Under slitlamp observation over a period of three or four weeks the opacity took on a more granular appearance, while the edema subsided. If the lesion was severe, the opacity would remain permanent, otherwise it would diminish gradually and in some cases disappear altogether with minimal or no scar formation.

Additional work in attempting to isolate virus from disciform corneas pointed up the similarity of the paucity of such isolations from animals as compared with poor results in humans. An LD_{50} dose for rabbit corneas of approximately 1,000 pock-forming units, in which only a benign dendritic of short duration occurred, suggests that poor isolation is due, in part, to low viral concentration in the inocula. Such inocula from humans is, at best, also greatly limited by the small amount of tissue available, and most likely, is obtained late when antibody has had a chance to accumulate in the cornea.

Various suggestions are offered as possible mechanisms that may be responsible for the production of a disciform keratitis. These are: (1) hypersensitivity of the cornea to herpes virus (as formerly suggested by Braley), (2) a possible association of corneal hypersensitivity to the virus with leukocyte infiltrates in the corneal stroma, (3) synergistic activity of virus with a secondary bacterial infection, (4) size of virus inoculum which may possibly include a viral toxin, (5) a mutant form of virus, (6) individual variation in host response.

Experimental ocular hypersensitivity: Some factors in the production of iridocyclitis in the rabbit by intravitreal injection of protein. Arthur M. Silverstein, Ph.D., and Lorenz E. Zimmerman, M.D., Armed Forces Institute of Pathology, Washington, D.C.

The work of others has demonstrated that injection of antigenic proteins into the rabbit vitreous can be accomplished without the production of early signs of inflammation or toxicity. In about one week, however, evidence of intraocular inflammation may be demonstrated. This response has been termed "primary anaphylactic iridocyclitis." It has been suggested that because of its anatomic, chemical, and physiologic peculiarities, the vitreous provides a depot from which the antigen is absorbed very slowly.

Experiments in our laboratory have shown that injections of more than 0.5 mg. of crystalline egg albumin into the rabbit vitreous will invariably produce an iridocyclitis after an incubation period of about seven days during which time the eye remains uninflamed. As the dose is reduced, the percentage of animals which respond with a "primary anaphylactic iridocyclitis" falls. When a dose of 0.1 mg. or less is employed, no ocular response is obtained. If, however, this 0.1 mg. dose is injected into one eye simultaneously with a large dose (1.0 mg.) into the other eye, both eyes respond with a typical iridocyclitis after the customary interval of seven days.

When the second eye receives a large (1.0 mg.) dose one to three days after a small dose (0.1 mg.) is injected into the first eye, the incubation period is reduced, both eyes responding with an iridocyclitis five or six days after the second injection. When, however, the second eye is injected with the large dose (1.0 mg.) six or more days after the first eye has received the small dose (0.1 mg.), only the second eye responds, and the incubation period is reduced to four or five days.

These observations, together with other immunologic data, indicate that intravitreal injections of crystalline egg albumin in the dosages employed (0.1 to 2.0 mg) will be absorbed into the circulation and will initiate antibody production. The failure of the small (0.1 mg.) dose by itself to elicit the "primary anaphylactic iridocyclitis" may be due to its inadequacy in stimulating sufficient humoral antibody to support the hypersensitivity reaction. Additional quantitative and temporal aspects of this response, including histopathologic observations, will be discussed.

Morphologic and functional development of the cornea. George K. Smelser, Ph.D., and Victoria Ozanics, M.S., Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York.

The formation of the fibrous components of the cornea by the cells of the loose mesenchymal primitive cornea is described with reference to

the time and location of their first appearance. Fibrillogenesis is correlated with the distribution of the sulfated mucopolysaccharides and metachromatic materials in the connective tissue ground substance. The embryonic mucopolysaccharides shown by their metachromatic reaction are attacked by hyaluronidase indicating that keratosulfate is found only in the late developmental stages and in the adult. Both fibrillogenesis and formation of the ground substance occur almost exclusively in the posterior lamellae in the embryo and fetus. The differentiation of these corneal components is contrasted with those of the sclera. The embryonic cornea possesses, as does the adult, marked hydrophilic properties. The development of this characteristic has been correlated with the degree of corneal hydration and the development of transparency. Embryonic corneas are more hydrophilic than other connective tissues at a very early stage of differentiation. The metabolic mechanism for controlling corneal hydration is also established in the very young and undifferentiated cornea.

The cornea in keeping with other embryonic tissues is translucent but not transparent. The differentiation toward transparency of the cornea is contrasted with the development of opacity in the sclera. Transparency develops slowly with the formation of more and more of the fibrous lamellae, metachromatic mucoid constituents of the ground substance, and gradual dehydration. Transparency, equivalent to that of the adult, is not achieved until all of the aspects of corneal structure and function which were investigated had essentially reached the adult condition.

Some aspects of the chemical composition of the aqueous humour and plasma of the smooth dogfish. William Stone, Jr., M.D., and Russell F. Doolittle, M.A., Ophthalmic Plastics Laboratory, Massachusetts Eye and Ear Infirmary, Boston.

A comparative chemical analysis of the aqueous humour and blood plasma of the smooth dogfish was undertaken. The elasmobranch fishes were chosen since the osmo-regulation of this class of organisms is very dependent on the retention of great quantities of certain organic compounds, namely—urea (elasmobranch blood urea approximately 100 times higher than human blood urea), and trimethylamine oxide. Inasmuch as urea in mammals does not reach a diffusion equilibrium between plasma and aqueous, it was theorized that a large absolute concentration gradient should exist across the elasmobranch "aqueous barrier". Consequently, factors tending to permit any such osmotic differential should be proportionately exaggerated. It was established in this study that a large concentration drop does exist for both urea and trimethylamine oxide. Therefore, in addition to urea and trimethylamine oxide, certain constituents which were known to be in either excess or deficit in many mammalian aqueous humours were measured. The ascorbic acid content of the aqueous was determined to be higher than in the plasma. Total CO_2 was also much higher, and preliminary pH studies show the pH to be considerably more alkaline. The gross concentration of free amino acids was shown to be much lower in the aqueous than in the plasma.

Electron microscopy of iris pigment granules (epithelial and stromal) in man and Rhesus monkey. A. J. Tousimis, M.S., and Ben S. Fine, M.D., Armed Forces Institute of Pathology, Washington, D.C.

Pigment granules of the iris epithelium and stroma (human and Rhesus monkey) were studied, measured, and compared, utilizing preparations made in two different ways. One type of preparation consisted of ultrathin sections of osmium tetroxide-fixed irises in which the pigment granules could be studied in situ. The other represented a concentration of pigment granules obtained by means of standard differential centrifugation procedures using dissected and homogenized stroma and epithelium. All preparations were studied with the electron microscope. Measurements of the pigment granules in the sectioned material were in general agreement with those made from the concentrated preparations.

In sections, considerable variation in size of the intracytoplasmic stromal granules was found in the human iris, the granules ranging from 0.1 to 0.6 microns in the minor axis and from 0.3 to 1.5 microns in the major. The granules of the pigment epithelium were found to be much more spherical and constant in size, usually measuring about 0.7 microns in diameter. The stromal granules in the Rhesus monkey iris were finer and more uniform in size and shape than were those in the brown human iris. Granules of the Rhesus monkey iris epithelium were similar to those of the human iris epithelium.

The effect on pigment granules of certain "bleaching agents" in general histologic use has also been studied with the electron microscope, utilizing isolated granules. The results of these studies and observations on the internal structure of pigment granules as revealed by ultrathin sections will also be presented.

Cells of the vitreous body: Cinephotomicrographic studies on the cells of the cortical layer of the vitreous body. L. Toth, Ph.D., E. A. Balazs, M.D., and E. A. Eckl, B.A. Retina Foundation, Department of Ophthalmology, Massachusetts Eye and Ear Infirmary and Harvard Medical School, Boston.

Cells taken from beneath the surface of bovine and rabbit vitreous body are photographed through a phase-contrast microscope. Slow movements are recorded with time-lapse equipment. In situ the cells are spherical, rotate slowly and

reveal cytoplasmic inclusions of various kinds.

If the cells find support on a solid surface such as a cover glass, pseudopodialike cytoplasmic extrusions may appear, followed by elongation of the cell. This type of cell may resemble fibroblasts with vigorously coiling and straightening mitochondria.

If there has been liquefaction of the vitreous gel within the eyeball or if it is facilitated by such means as collagenase treatment, some of the spherical cells become flattened and resemble glial cells as they appear in tissue culture.

The formation and extrusion of bubbles is seen in both bovine and rabbit cells, and gives one the impression of a secretory activity.

When the eyeball is stored for longer than one hour after the death of the animal, a morphologic change develops which cannot be seen in fresh preparations. A layer of bubbles forms above the cells while the latter still exhibit vigorous movements. In mounting the specimen, the layer of these bubbles folds in accordionlike pleats which give an impression of parallel running fibers. After six to eight hours of storage, the bubbles disappear and only a membranelike structure with the folds and cells remains.

The cytoplasmic inclusions of the living cells rapidly take up neutral red and sometimes release it with or without signs of cell degeneration. The granules stain orthochromatically with azure A and give a positive periodic acid-Schiff reaction.

Electron microscope study on normal lens fibers. Theodor Wanko, M.D., and Mary Ann Gavin, M.S., Ophthalmology Branch, National Institute of Neurological Diseases and Blindness, Bethesda, Maryland.

Cortical lens fibers were studied from specimens of normal adult rats and rabbits. Lens fibers appear as prismatically shaped cells, separated from each other by a less opaque space between two dense, cellular membranes. The fibers frequently interdigitate with one another. Their boundary towards the capsule is formed solely by the individual cell membrane.

Within the cytoplasm of fibers the following elements are discernible: mitochondria; endoplasmic reticulum of the granular type; clusters of dense granules which lack visible attachment to membranes; aggregrates of small vesicular profiles with varying densities.

In the bow region of the lens fibers the nuclei can be distinguished from the surrounding cytoplasm. They contain one or two distinct nucleoli, which appear as congregations of dense granular particles.

The foregoing components are embedded in a mass of closely packed, filamentous structures, which are characterized by a diameter of about 100 Å, low density to electrons, and indefinite length. They are distributed at random throughout the entire cytoplasm.

Transport of neutral red by injury-activated corneal stromal cells. Virginia Weimar, Ph.D., Department of Ophthalmology Research, College of Physicians and Surgeons, Columbia University, New York.

It has been observed previously that corneal stromal cells in normal corneas do not take up the vital dye neutral red. However, within 12 hours after wounding, all the corneal stromal cells are activated to transport this dye in large quantities. The mechanism by which these activated corneal stromal cells actually take up the dye is unknown, but this phenomenon has provided a useful tool for detecting the influence of various chemicals on the early steps of wound healing.

A procedure was developed for producing in vitro the activation of the corneal stromal cells to transport the vital dye neutral red under controlled and reproducible conditions. A simple method of extracting the dye from the cornea has made it possible to measure quantitatively the amount of dye taken up by the activated corneal stromal cells.

From these studies in vitro it has been established that there are at least two steps involved in the uptake of neutral red by corneal stromal cells: (1) activation of the corneal stromal cells to take up the dye, and (2) uptake of the dye by the activated cells.

The epithelium plays an essential role in the activation of the corneal stromal cells. If the epithelium is removed without trauma of any kind, the corneal stromal cells do not become activated and do not take up any dye. Crushing the epithelium, however, leads to activation of the corneal stromal cells within one hour after injury.

Proteolytic activity in the wounded corneal epithelium appears to be necessary for the development of the neutral red reaction. Specific protease inhibitor prevents the activation of the corneal stromal cells, but it is effective only in the presence of intact epithelium. If the epithelium is crushed, the protease inhibitor is no longer effective. Crushing the epithelial cells apparently releases an unknown factor (or factors) which acts directly on the corneal stromal cells to activate them, thus by-passing the step of proteolytic activity.

Trypsin is an effective activator of the neutral red reaction; but it produces activation only in the presence, but not in the absence, of epithelium.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

CHICAGO OPHTHALMOLOGICAL SOCIETY

May 19, 1958

DR. FRANK W. NEWELL, *President*

VITREOUS SURGERY

DR. DONALD SHAFER of New York spoke on the latest developments in implantation of vitreous. In a series of over 100 secondary operations performed for retinal detachment a cure rate of about 60 percent was obtained. Dr. Shafer emphasized that one must choose the appropriate operation for a specific detachment and not look upon vitreous implantation as the method of choice in all eyes with this tragic disease. Furthermore, the sine qua non is still closure of all retinal tears. A preliminary presentation was also made on the bacteriology of the vitreous. Various organisms were grown on blood-agar plates and a drop of vitreous and of several antibiotics placed on the plates. In almost every case the zone of inhibition of growth was largest around the vitreous. Dr. Shafer concluded by saying that this was still an experimental procedure with many problems of the fundamental nature of the material implanted still to be studied.

Discussion

Question. What would you consider the minimum amount of vitreous you should have on hand at the start of an operation?

Answer. The actual answer depends upon your appraisal of the volume of subretinal fluid you are going to lose. But tentatively, I like to have three cc., if possible four cc., in my syringe when I am implanting. Sometimes an eye will take three cc., but most of them will take about one cc. to one and a half cc. of vitreous.

Question. Can you use an eye for a corneal transplant after the vitreous has been removed?

Answer. That depends on the corneal surgeon. We have done it the other way around. We have taken the cornea first, then taken the vitreous. Personally, I don't know of any objection but we have not done it, for our corneal men became so upset about somebody tampering with their donor eye that we have never done it. What we do, after the button is cut out, is to displace the lens down, go right through the donor site, and aspirate out the vitreous.

Question. Is the incision the same in the aphakic eye?

Answer. Yes, the incision is the same in the aphakic eye. On that point, we thought we would certainly get vitreous in the anterior chamber, glaucoma, and so on. We haven't. I believe that is because it is fluid vitreous—that is, vitreous aspirate. We see free vitreous in the anterior chamber many times after discission and we don't get cloudy corneas or any glaucoma. I believe it is the hyaloid rather than the contents of the hyaloid that causes glaucoma and corneal opacities.

Question. Is it necessary to use the 18-gauge needle rather than a smaller needle?

Answer. Some men have been using smaller needles; Dr. Pischel and Dr. Clark of New York have. I've used an 18-gauge needle to implant the vitreous because that was the size of the needle that we used to withdraw it from the donor eye. I feel that if there is some structural remnant present why break it down any further. I think if you are going to do it, you might as well stay up with an 18-gauge or drop down to a 25-gauge. Otherwise the vitreous will seep through just a straight puncture hole.

Question. Do you try and localize your site of injection at the area of detachment or would you rather go some other place?

Answer. You can go anywhere. For a while, statistically it seemed to look as though the results were a little better if the vitreous

implanting site was 180 degrees from the major tear but it doesn't now. I use as an implanting site, supratemporally or supranasally so you don't have to work over the nose if that sclera is good. If the sclera has been burnt up by previous surgery, then it is better to shift nasally so that your sutures are in good sclera.

Question. At what temperature is the vitreous injected into the eye?

Answer. The vitreous is at 40°F. when it comes to the operating room. We leave it in the rubber-stoppered vial and the vial in 70-percent alcohol for a half an hour. The alcohol is, of course, at room temperature, roughly 72°F. or so, so the actual implanting temperature is somewhere between 40°F. and 72°F.

Question. Within what period after enucleation or after death must the vitreous be withdrawn?

Answer. Apparently the requirements are about the same as for the cornea. Certainly, if the eye is enucleated within 12 hours after death, we seem to have no difficulty with the use of the vitreous. Very often the vitreous is not removed from the eye itself for 72 hours. Most of our vitreous comes from eyes that have been too long enroute so that the cornea is no longer suitable and we find that the vitreous still seems satisfactory.

Question. Is there any advantage to the use of vitreous rather than saline or air? Have you studied the fate of the implanted vitreous?

Answer. Apparently there is. We all know that air certainly soon disappears after being implanted. We believe saline does too but I believe vitreous does not. We tried dyeing it and we found, of course, when we dyed it that all it did was mix with the vitreous the patient still had and we just ended with one homogenous dyed vitreous, which is of no advantage.

We considered trying to tag it with radioactive material and, after correspondence back and forth with Cogan on his trying to tag radioactive corneas for the fate of the implanted cornea in a corneal transplant, we felt that this would not be successful. That is why we are in such a position that one radioactive specialist says:

"The only way you can do it is feed radioactive food to pregnant rabbits then, when the baby rabbits are born, they will be radioactive. Let them become adults, for their vitreous is so little, and then you can get a little bit of their radioactive vitreous. Plant it in another rabbit that is not radioactive and that should do it."

But we have not done all this yet.

As far as the pure surgical advantage at the operating table, air versus saline versus vitreous, I don't think there is any advantage. Maybe air has a little advantage at the moment. I think an air bubble will tamponade a larger tear than saline or vitreous. But I believe, once it is present, the vitreous seems to remain and I know air does not and I don't think saline does. I believe it is the long-term effect that is the advantage of vitreous. There is also apparently a softening effect on some of the fixed retinas and on the vitreous bands. Again I say that it is not dependable for I have seen a mass of vitreous retraction after I have done a vitreous implant and I've seen it after resections and also after diathermy alone. So, it is not a sure cure for vitreous bands or fixed retinas, but—in enough cases—the men who are doing them seem to feel that it is a factor and that I believe is the advantage of vitreous.

Residents' program

The remainder of the evening was devoted to the first annual residents' program. Six papers were presented and, of these, two were thought to be outstanding by the judges. Dr. James McCarthy of Cook County Hospital presented "Experimental Nocardia asteroides infection of the rabbit eye." He implanted Nocardia asteroides into the anterior chamber and beneath the conjunctiva of the rabbit eye. Microabscesses developed which gradually involved the remainder of the globe. Extraocular metastases may occur. Dr. Joseph Hatch of the Illinois

Eye and Ear Infirmary discussed "Ochronosis and its ocular findings." A case history was presented and the X-ray, laboratory, and other diagnostic features elaborated.

David Shoch,
Recording Secretary.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

February 3, 1958

Dr. Harvey E. Thorpe, *President*

Lacrimal air anomalies

Dr. Jesse M. Levitt said that lacrimal air anomalies, which are rarely observed, are caused by defective valvular function of the nasolacrimal duct, usually on a congenital basis, permitting abnormal passage of air, as well as nasal secretions, into the lacrimal system. There is no mechanical obstruction to the flow of tears but a tendency toward impaired drainage from malfunction of the lacrimal sac and infection from the nasal secretion.

Two unusual cases were presented. The first was in a 13-year-old girl who complained of inconstant tearing of the right eye. There was no history of eye infection or trauma or nasal disease. Palpation over the right lacrimal sac disclosed a tiny pneumatocele, with crepitation loud enough to be heard a few feet away. Fluorescein solution dropped into the eye was not transmitted into the nose but irrigation through the lower canaliculus was exceptionally free into the nose. When she blew her nose forcibly with the nostrils occluded, the pneumatocele sometimes enlarged.

Dacryocystography showed a diverticulum of the lacrimal sac and markedly deficient drainage of tears, without any mechanical obstruction to their flow. It was felt that dacryocystorhinostomy with excision of the diverticulum provided the best chance of cure.

The second case was that of a 36-year-old woman who complained of tearing of the left eye of three months' duration. When she blew her nose air bubbles came through the lower lacrimal punctum. Here again fluorescein solution dropped into the eye was not transmitted to the nose, while irrigation through the lower punctum was free into the nose. Dacryocystography revealed a patent lacrimal tract, a dilated lacrimal sac and nasolacrimal duct, and deficient drainage of tears.

Treatment consisted of irrigation of the lacrimal tract with a solution of Chloromycetin, local use of the same solution, and instruction to blow the nose gently, one nostril at a time, at the same time pressing firmly on the lacrimal sac area. This effected a complete cure, at least up to the present, a period of four months.

Clinical significance of intrascleral nerve loops

Dr. Dewey Katz reviewed the literature and histology of intrascleral nerve loops, and stated that Axenfeld first called attention to them in 1893. Intrascleral nerve loops have been mistaken for foreign bodies and tumors: hence the clinical importance of identifying them. They are found in the plane of a rectus muscle, three or four mm. from the limbus, protruding slightly, with the appearance of a grayish-white, gelatinous spot, over which the conjunctiva moves freely. Close observation shows next to it a small blood vessel, an anterior ciliary artery, passing through the same emissarium. Grasping the nerve loop with a forceps induces pain even after the instillation of a local anesthetic. The loops are surprisingly common. Dr. Katz observed eight in the course of a year.

Discussion. Dr. Gartner commented that intrascleral nerve loops are not infrequently seen in the laboratory when the eyes are sectioned in the horizontal plane.

Prof. A. Fuchs remarked that intrascleral nerve loops have often been observed with the aid of simple loupes.

Gonioscopy in the Management of Certain Secondary Glaucomas

Dr. Adolph Posner divided secondary glaucoma into three categories:

The first group, in which the angle closure mechanism operates, includes: (1) seclusion of the pupil with iris bombé, (2) anterior dislocation of the lens, (3) vitreous-block glaucoma, (4) air-block glaucoma, (5) malignant glaucoma.

The second group, in which the formation of peripheral anterior synechias is the primary cause, consists of: (1) glaucoma following a flat anterior chamber, (2) glaucoma which may develop late in the course of uveitis, (3) essential atrophy of the iris, (4) rubeosis iridis, (5) posterior dislocation of the lens, (6) tumors of the ciliary body and iris, (7) burns and severe inflammations involving the chamber angle.

The third group consists of miscellaneous types of glaucoma in which the mechanism is not well understood: (1) glaucoma occurring early in uveitis, (2) glaucomatocyclitic crises, (3) glaucoma capsulare, (4) pigmentary glaucoma.

Dr. Posner stressed the importance of gonioscopy in differentiating glaucoma secondary to iridocyclitis from acute angle-closure glaucoma. In angle-closure glaucoma the angle is found to be closed and in the fellow eye the angle is usually of the narrow type, though open. In secondary glaucoma the angle usually is open, except possibly for a few localized synechias, which cannot account for the elevated tension. Angle-closure glaucoma may be asymptomatic and without gonioscopy could easily be mistaken for simple glaucoma.

There is a vast difference clinically between acute noncongestive and congestive glaucoma. The acute congestive attack is considered an endophthalmitis produced by protein-split products. There is a strangulation of the arterioles which supply the iris and ciliary body, resulting in tissue necrosis. The necrotic uveal tissue acts as a foreign protein giving rise to inflammation resembling iritis or endophthalmitis. During the hypertensive phase the eye shows corneal edema, shallow anterior chamber, closed angle, aqueous flare and cells, posterior synechias, and bits of pigmented iris tissue floating in the aqueous (iridoschisis).

The tell-tale of a past attack of acute congestive glaucoma is a triad of signs: (1) sector-shaped atrophy of the iris resulting usually in a distorted pupil, (2) pigmented particles on the posterior cornea and anterior surface of the lens, (3) capsular and subcapsular lens opacities.

Sympathetic Ophthalmia without History of Injury

Dr. S. Lawrence Samuels presented an interesting case of sympathetic ophthalmia which was etiologically a puzzle that the pathologic examination was able to solve. A 64-year-old man gave a history of sudden marked diminution of vision in the left eye in November, 1949, with no pain and no apparent injury. The diagnosis was vitreous hemorrhage and there was no sign of anterior segment inflammation or external injury. Within a few months the eye was totally blind. He continued to have left frontal headaches and short bouts of redness and pain in the left eye but did not seek medical advice again until four years later when enucleation was advised and refused. In May, 1956, the right eye became very red and painful and soft with an acute uveitis and marked diminution in vision. Intensive treatment controlled the inflammation. The left eye was shrunken, white, soft, with a cloudy cornea, and an indentation of the external scleral sulcus in the upper half. This time he consented to enucleation when it was pointed out that the inflammation in the seeing eye could be related to the blind atrophic eye.

When this man was first seen, I thought that here, at long last, is an authentic case of sympathetizing inflammation with no history or evidence of a perforating injury or ruptured globe, no intraocular surgery, perforating ulcer, or necrotic malignant melanoma.

The histologic examination confirmed the suspicion of sympathetic ophthalmia.

The history, however, in spite of protestations to the contrary by a well-meaning elderly man and his son and the report of the first doctor to see him at the onset of his illness, must be discounted in the light of definite evidence of an incised wound and complete absence of lens remnants in all the sections studied.

At the limbus on both sides, running through the stroma to the anterior chamber, is a well organized scar with a marked indentation between the cornea and sclera at the external scleral sulcus. The process is one of a diffuse uveal inflammation of the granulomatous type involving mainly the posterior parts of the choroid and iris and the vascular layer of the ciliary body. The involvement of the sympathizing eye did not occur until at least six and one-half years after the first recorded symptoms in the exciting eye. (This illustrates that an old atrophic eye, shrunken and quiescent, and showing no inflammatory activity, may start up an acute inflammation in the fellow eye years after the initial injury.)

In conclusion Dr. Samuels stressed the following points: (1) Don't trust the history, (2) enucleate all blind, shrunken eyes, (3) don't throw away any enucleated eyes; have all eyes sectioned and studied, (4) continue steroid therapy and periodic check-ups for a long time in cases of sympathetic ophthalmia.

Discussion. Prof. A. Fuchs deduced from the appearance of the globe that the patient had had an operation rather than an injury, and found it hard to understand that the patient should not have known this. He commented that, while cortisone may be a great help, its use should not make one too optimistic. He recently saw two cases that became entirely blind despite cortisone. He also noted that sympathetic ophthalmia is not so rare as has come to be believed within recent years.

Jesse M. Levitt,
Recording Secretary.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

440th Meeting, January 15, 1958

Virgil G. Casten, M.D., *Presiding*

Problems in glaucoma, I

Dr. W. Morton Grant, Boston: In the majority of cases of open-angle glaucoma we can see nothing wrong. We have very little to go on. We study enucleated eyes and hope to find the answer but, in spite of over 75 years of investigation, very little has been learned.

Part of the problem in investigating enucleated eyes for the mechanism of open-angle glaucoma is that open-angle glaucoma has only been well recognized in recent years. It has only been in the last 10 years that the classification of the various glaucomas has been clarified. Rarely does an eye with primary open-angle glaucoma reach the pathologist. The eyes often go quietly blind, cause no pain, and are rarely removed. So very few have been studied and none have been well studied clinically and then pathologically.

Supposing we did have several such eyes, would we then find the answer by sectioning them and studying them? I don't think so. The changes in these eyes must be rather subtle—the matter of very, very small changes in the diameters of channels.

We really have to know well all the characteristics of the normal eye in order to make a comparison of such minute details. To determine these necessary anatomic facts we use microdissection methods. By this means it has been determined that about 75 percent of the resistance to outflow is present in the trabecular meshwork. There is evidence that, functionally, this resistance is varied by such forces as the miotic drugs exert on the ciliary muscle.

Assuming we have established adequately with normal eyes all the characteristics necessary, we then examine an open-angle glau-

coma eye and expect the resistance to outflow to be greater even after enucleation. What has brought about this change—what components of the trabecular meshwork have changed?

By simply looking at the tissues it would be very difficult to determine whether the pores were significantly altered in size. The process of fixing the tissue for microscopic examination always alters the pore sizes. Fixation in formaldehyde obstructs the pores and an artefact is introduced. Let's leave this problem for a time.

In examination of the angle in sections we sometimes see strands of tissue that are not the corneoscleral meshwork itself but are really closer to the anterior chamber. When you look at the angle gonioscopically, you are often aware that overlying the corneoscleral trabecular meshwork is a whole network of other fibers, the uveal meshwork. We tend to ignore this even though in some adult eyes the meshwork is developed enough so that it really obscures the whole ciliary muscle and the scleral spur and is overlying part of the corneoscleral meshwork.

Sometimes in an open-angle glaucomatous eye this structure is so dense as to make us wonder if this could be responsible for the obstruction to flow.

We have similar thoughts when we consider the infant eye and in what manner the development of this part of the meshwork is tied up with congenital glaucoma. In the infant eye the corneoscleral trabecular meshwork, the scleral spur, and the ciliary muscle can be seen more clearly than in the adult eye. On a plane but separated from these structures is a gelatinous tissue, quite uniform and extending from the iris periphery across the angle structures up to the region of Schwalbe's line. As time goes on little strands of pigment, in the brown eye, come up from the iris periphery onto this sort of embryonic uveal meshwork and in time it may become deeply pigmented, shrunken down, and coarsened in appearance. In the adult eye instead of being separated to a considerable depth from the corneoscleral meshwork it is applied right on the surface of these structures.

When you have two eyes in the same individual, one eye glaucomatous and the other not or much less so, in a few instances it has been noticed in the glaucomatous eye that this tissue veils the underlying scleral spur and corneoscleral trabecular meshwork more than in the normal eye. This makes you wonder if this structure is responsible for interfering with the outflow in these cases of congenital glaucoma.

Problems in glaucoma, II

Dr. Paul A. Chandler, Boston: The medical and surgical treatment I give to patients with exfoliation of lens capsule and secondary glaucoma is no different from that given to other cases of open-angle glaucoma, except that I think I might remove the lens a little earlier in the cases of exfoliation and glaucoma. There is, however, no clear-cut evidence that removing the lens halts the process. There is some question if this material come from the lens or some other source.

When there is subluxation of the lens from injury and the lens is tilted forward and producing a closure on one side, it is well to do nothing surgically until the immediate effects of the injury have passed and the eye is quiet. This may be some weeks and the closure will almost certainly be permanent, with peripheral synechias. Removal of the lens would not open the portion of the angle closed by the synechias; on the other hand, removal of the lens in such cases might cause massive vitreous loss. If the lens is not opaque it would be of more use to the patient than an aphakic eye would be. If the lens is opaque and some months have passed and if the patient is over 35 years of age, then the lens may be removed without too great a danger of vitreous loss. For younger patients in this group it is better to get rid of the lens by discission.

Angle-closure glaucoma caused by tilting

of the lens may be held in check for an indefinite period with the help of miotics. If the lens allows useful vision and miotics fail, I would be inclined to do a filtering operation rather than remove the lens. If the lens is clear or almost clear, it is better in than out from a visual standpoint.

In Marfan's syndrome when the lens goes into the anterior chamber, there may be glaucoma due to the pupillary block. I have not been able to relieve this glaucoma by dilating the pupil. Removal of the lens in these cases when the tension is sky high is a risky procedure. I believe the best thing is to make a hole in the iris, either a little iridotomy or a transfixation, to relieve the block and the glaucoma. When the iris drops back and when the eye is soft then the lens can be removed and there is a chance of fewer complications. It is a serious situation but there isn't much choice when the lens is in the anterior chamber.

Now coming to cataracts—we all know that a swollen lens, immature, mature, or hypermature, can cause a shallow chamber and typical angle-closure glaucoma. Here we should first try to reduce the tension by medical means and then remove the lens. An iridectomy is not indicated in these cases. There is a good chance that a complete cure can be effected by removal of the lens.

With the hypermature cataract with deep chamber and wide angle, there is sometimes a glaucoma that is due to a block of the meshwork by macrophages. Medical treatment will not get these cells out of the meshwork. Lower the tension by the usual means, remove the lens, do not do an iridectomy.

When there are immature cataracts and open-angle glaucoma, there is reason to believe they are two separate diseases. In these cases it has been advocated to do a combined lens extraction and iridencleisis. This practice has never appealed to me on the grounds of theory and I have never employed it.

In young or middle-aged people with severe, medically uncontrolled open-angle glaucoma and a great degree of lens opacity, I do a filtering operation for the glaucoma and some months later remove the lens. In older people whose glaucoma is not too bad I have often removed just the lens and counted on medical treatment of the glaucoma after the lens extraction.

The secret of avoiding glaucoma following the discission of an intact lens is to open the capsule widely from top to bottom and from side to side. Then the lens cannot swell as a whole; the cortex will be extruded into the anterior chamber. The angle will never close from swollen lens if the capsule is opened widely. However, if this glaucoma does occur, do not use medical treatment except for the preoperative use of Diamox. Remove the lens.

Pupillary block is a rare form of glaucoma in aphakia. You can get it from an adhesion of the intact hyaloid to the iris. It is practically unknown in a patient that has had a full iridectomy, unless the patient has had a flat chamber. It is most common in round-pupil extractions where the tiny peripheral iridotomy or iridectomy is closed by adhesion of the wound to an inflammatory membrane.

Sometimes, with a round pupil extraction, we see a block by a mushroom of vitreous coming forward into the anterior chamber. When this happens, it is always wise to make an opening in the periphery of the iris before irreversible damage is done to the cornea by contact.

In glaucoma secondary to inflammation of the eye the meshwork is presumably blocked with cells and proteins. Here is a wonderful case for Diamox. I used to puncture these eyes right and left, now that is very rarely necessary for most of them are controlled with Diamox. I treat the inflammation in the usual way.

Charles Snyder,
Recorder.

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INTERNATIONAL COUNCIL OF OPHTHALMOLOGY AND INTERNATIONAL FEDERATION OF OPHTHALMOLOGICAL SOCIETIES

A meeting of the International Council of Ophthalmology was held during the XVIII International Congress of Ophthalmology in Brussels, on September 6, 1958.

The following is a brief résumé of the more important business.

1. *Report of the treasurer.* The financial position of the council was reported to be satisfactory. The contribution to the Council for International Organizations of Medical Sciences came under review. Hitherto the CIOMS received 100 U. S. dollars annually from the council and each international congress paid 500 dollars to the same body. In view of the increased subsidy desired by the CIOMS, the council undertook to pay 300

U. S. dollars annually and hoped to recoup itself to the extent of 500 dollars from each international congress. Great regret was expressed at the resignation of Amsler from the post of treasurer which he had filled for eight years with much distinction.

2. It was considered desirable to give more publicity to the *Report on Ophthalmological Education* throughout the world, presented to the council in 1954. This report will be published in the *British Journal of Ophthalmology* and a reprint will be sent to all universities with a faculty of medicine.

3. It was decided that the recently formed European Society of Ophthalmology would be represented on the international council; François was accepted as the delegate. This society, which has been joined by all countries in Europe except Bulgaria and Russia, will hold its first congress in Athens in April, 1960.

4. The *Index Ophthalmologicus* (1958) was distributed gratuitously to all members attending the XVIII International Congress at Brussels. Further copies of this volume are obtainable through Dr. A. C. Copper (Coehoornsingel 42, Zutphen, Holland).

5. *The Multilingual Ophthalmological Dictionary*, published under the auspices of the council, will be on sale early in 1959; the publisher is S. Karger, Basel.

A meeting of the International Federation of Ophthalmological Societies was held in Brussels on September 8, 1958.

The recommendations of the international council were in general approved. The following is a brief résumé of the other business.

1. It was decided that the subscriptions paid by affiliated societies should remain at the present figure of one-half Swiss franc per member per annum.

2. The new statutes put forward in the international congress of 1954 in Montreal were adopted with the modification that the national committee organizing the international congress would have the right to refuse to accept communications submitted to it in order that the number of such communications could be reduced and their standard raised.

3. Two reports already passed by the international council were accepted by the federation. In the testing of the color sense in transport workers by isochromatic plates, the Swedish test, Boström (B II 1950) was admitted, as well as the test of Ishihara. A report on visual safety on trunk roads concluded that trees bordering them or advertisements, if not too numerous, gave rise to no danger from the visual point of view.

4. The ophthalmological societies of Burma and Tunis were accepted as members of the international federation.

5. It was decided that the XIX International Congress should be held in India at New Delhi in 1962.

6. The new international council. Officebearers to the international council and the replacements of five of the 10 ordinary members were elected to hold office during the next four years. The new council consists of the following: Sir Stewart Duke-Elder (president); C. Berens (vice-president); E. Hartmann (secretary); B. Streiff (treasurer).

Members: Arruga (Barcelona), Palomino Dena (Mexico), Espildora Luque (Santiago, Chile), Lyle (London), Melanowski (Warsaw), MacDonald (Toronto), Müller (Bonn), Paufique (Paris), Uyemura (Tokyo), Weve (Utrecht).

Ex officio. Bietti (International Association for the Prevention of Blindness); Franceschetti (International Organization Against Trachoma); Payne (Pan-American Association of Ophthalmology); François (Pan-European Society of Ophthalmology); Coppez (president of the last congress); the president of the next congress; Copper (secretary for the *Index Ophthalmologicus*).

Stewart Duke-Elder.

OPHTHALMOLOGIC GOOD-WILL TOUR TO ASIA

Following the XVIII International Congress of Ophthalmology, a group of seven United States ophthalmologists, accompanied by three of their wives, left on a good-will tour of Asia. This tour was under the auspices of the newly formed Asia-Pacific Academy of Ophthalmology.

This academy, modeled after the Pan-American Association of Ophthalmology and the European Society of Ophthalmology, was created to extend ophthalmologic knowledge and to advance the art and science of ophthalmology and related sciences in Eastern Asia, the Western and Central Pacific, and Oceania.

Advance arrangements had been made and a mutually convenient schedule had been established with officers of ophthalmic societies in Pakistan, India, Ceylon, Thailand, Hong Kong, and the Philippine Islands.

Members of the group included Dr. and Mrs. A. Benedict Rizzuti of Brooklyn, New York, Dr. H. Saul Sugar of Detroit, Michigan, Dr. Richard A. Perritt of Chicago, Illinois, Dr. and Mrs. Charles S. McWilliam of Newburgh, New York, Dr. Harold E. Hunt and Dr. Thomas E. Hunt, Sr., of Paris, Texas, Dr. and Mrs. William John Holmes, of Honolulu, Hawaii. Joining the group in various cities in Asia were Dr. and Mrs. Maurice Rothberg of Fort Wayne, Indiana, Dr. and Mrs. Ronald Lowe of Melbourne, Australia, Dr. and Mrs. George O. Zugsmith of San Pedro, California, Dr. and Mrs. Henry R. Nesburn of Los Angeles, California, and Dr. and Mrs. G. H. Levien of Hamilton, New Zealand.

In keeping with the aims of the Asia-Pacific Academy of Ophthalmology and to promote scientific communication between the visiting ophthalmologists and their overseas colleagues the program of the meetings consisted of lectures, demonstrations, exhibits, panel discussions, clinics, and films. Some of the papers of special interest included "Pupil block glaucoma in phakic and aphakic eyes" and "Gonioscopy and angle-closure glaucoma," Dr. Sugar; "Acute glaucoma requiring lens extraction," Dr. Lowe; "Recent advances in keratoplasty" and "New techniques in cataract surgery," Dr. Rizzuti. Popular moving-picture films were those on keratoplasty and new techniques in cataract surgery by Dr. Rizzuti; on acrylic implants by Dr. Perritt; and on surgery of the superior oblique muscle presented by Dr. Harold Hunt.

The panel discussions were especially well received because they brought out differences of opinion among the panelists. The entire group participated in these.

In Karachi, the group was met at the airport by the officers of the local eye society. The day after our arrival, we were taken on a tour of three local hospitals. We were especially impressed by the work of Dr. M. H. Rizvi and his associates (all of them under 29 years of age) on keratoplasty. Through the courtesy of Dr. A. D. Minhas and Prof. M. A. Shah we saw trachoma in all of its stages. We also learned to appreciate the hardships and difficulties, trials and accomplishments of ophthalmic surgeons in this new country. We received our first introduction to Asian hospitality at a lavish buffet dinner given in our honor by the Pakistan Ophthalmologic Society at the Beach Luxury Hotel of Karachi.

In New Delhi, Dr. N. S. Jain, president, and M. N. Razdon, secretary, of the Delhi Ophthalmological Society arranged our lectures and visits to the Irwin, Schroff, Sir Ganga Ram, and Jeewan Eye Hospitals. Following our lectures we were fortunate in meeting Dr. S. P. Schroff, founder of the famed Schroff Eye Hospital and dean of Delhi ophthalmologists. The inscription, "Live to Serve," on the stationery of this great, 84-year-old, surgeon provides an insight into his philosophy.

In New Delhi we were invited to represent the American Medical Association at the foundation stone laying ceremony of the

Indian Medical Association's new building. On this occasion, some of us were privileged to meet with leaders of the Indian medical profession and with Dr. Rajendra Prasad, the president of India.

In Bombay, we were also met at the airport by Dr. Dhurandhar, president of the Bombay Ophthalmological Society, Dr. Y. K. C. Pandit, and others. Here again we were invited to visit several hospitals. Following our lectures, we were taken to a dinner party at the fashionable Wellington Sports Club.

In Madras, Dr. V. Rangachari had especially arranged a dance recital for us by one of South India's foremost exponents of ancient and religious dances. He had also given us a beautifully engraved silver platter, for presentation to the artist and her troupe, commemorating our visit to Madras.

At the Government Ophthalmic Hospital in Madras, we were especially delighted by the vast collection of clay models and glass eyes depicting various forms of ocular pathology frequently seen in the tropics.

In Aligarh, we were shown the facilities of the Muslim University Institute of Ophthalmology by Dr. B. R. Shukla and Dr. M. V. Gupta. Here we also were treated to a number of well-documented exhibits prepared under the inspiring leadership and guidance of Prof. F. C. Rodger. These included:

1. The Carr-Price reaction, by A. Fazal.
2. A dark adaptometer for use with the experimental animals, by F. C. Rodger.
3. The low viscosity nitrocellulose technique of embedding, adapted for hot countries, by F. C. Rodger.
4. Effect of all trans-isomer of vitamin A on hemeralopia, by H. Saidozzafar.
5. Demonstration of the rat visual pathway entirely sectioned, in one plane, by S. Hammed and K. Nath.
6. Pathologic slides: retinoblastoma in a man aged 48 and ossification and amyloidosis of the tarsal plate, by S. Hammed.
7. Trachoma inclusion bodies in epithelial scrapings, by K. C. Agarwal.
8. Fluorescent microscopy for vitamin A studies, by Dr. Agarwal and Dr. Leekha.
9. Techniques of tonometry and tonography with monkeys, by A. S. Grover.
10. Flim on the prevention of blindness in India.

In Aligarh, we were the luncheon guests of Col. B. H. Zaidi, vice chancellor of Muslim University and his charming, vivacious wife.

Wherever we went in India, we were besieged by phone calls, letters, wires, visits from patients who had heard of our arrival, and, on their own or accompanied by their physicians, sought consultation. Generally we found that we had only to verify the diagnosis and treatment as recommended by their own ophthalmologists.

In Ceylon, we were again met at the airport by the president, Dr. W. H. V. Ferninands, and other officers of the local ophthalmologic society. Our arrival in Colombo was timed to coincide with the First Annual Congress of the Ophthalmological Society of Ceylon. This society, formed in December, 1957, and numbering 30 members has already held six meetings, publishes a quarterly ophthalmic journal, *The Journal of the Jaffna Clinical Society*, and enjoys the use of the well-equipped Victoria Memorial Eye Hospital.

Here we were shown patients with xerosis, keratomalacia, and orbital tumors. The scientific exhibit prepared by the society consisted of selected ophthalmopathologic specimens, an exhibit on the role of eye flies in epidemic keratoconjunctivitis, and a rare case of Philopthalmus looss (a parasitic trematode found in birds) that had involved the conjunctiva.

We were pleased to hear that, unlike in other parts of Asia, trachoma in Ceylon was of little, if any, consequence. We were also impressed by several successful cases of keratoplasty performed by Dr. Sivasubramaniam and by an up-to-date textbook of ophthalmology which this young, energetic, capable surgeon had written for medical students.

At the hill station of Kandy, we watched and photographed elephants at work and cavorting in the river, and tasted and inhaled the fragrance of the varied spices of the

Orient at the beautiful botanical gardens.

Back in Colombo, the climax of our visit was the official dinner of the congress held at the Grand Oriental Hotel. This affair was attended by over 100 physicians and their wives, the largest medicosocial get-together in the history of Ceylon. Following a toast to Her Majesty the Queen of Sri Lanka (Queen Elizabeth) the remaining toasts emphasized how physicians the world over were anxious to share their knowledge to alleviate human suffering; and how through the exchange of medical information physicans of different countries get to know and respect one another.

Leaving Ceylon, we stopped in Calcutta. At the eye infirmary we were granted an opportunity to observe various types of eye surgery. Here we also had a glimpse of the devoted, fruitful, and far-reaching work done by the West Bengal Association for the Prevention of Blindness.

Even though we arrived in Bangkok, Thailand, two days behind schedule, ophthalmologists from both Siriraj and Chulalongkorn hospitals gathered on short notice to meet with us. In order to spend more time with us, a large group of them saw us off at the airport at 2:00 A.M.!

In Hong Kong, Dr. G. C. Dansey Browning, vice chairman of the ophthalmologic society, and several other members and their wives welcomed us. Soon after our arrival they treated us to a Chinese Yum-cha (jasmine tea, long-life noodle, moon cake, and other delicacies). The same afternoon we were invited to a concert rehearsal of the Hong Kong Music Training Centre for the Blind.

This one-year-old organization, the only one of its kind in Southeast Asia, is sparked by the enthusiasm of Mrs. Renald Ching, whose capable and highly respected ophthalmologist husband was trained at the University of Chicago. Fifteen young school-age performers sang both English and Chinese songs as a choral group and played instrumental music as well. One of the highlights of our stay in Hong Kong was a visit to Hay Ling Chau. Here under the kindly and competent management and Christian compassion of Dr. N. D. Fraser is the model leprosarium of the world! Dr. Fraser's policy is built on voluntary segregation, compulsory treatment, and active participation by the patients in the work of keeping the colony going or in some form of occupational therapy.

At Hay Ling Chau, many of us for the first time examined, studied, and photographed the manifold ocular and systemic manifestations of leprosy.

Prior to our departure from Hong Kong, we were entertained at an elaborate, nine-course farewell dinner by the ophthalmologic society.

Upon our arrival in Manila, the pattern to which we had already become accustomed again repeated itself. Dr. Geminiano de Ocampo, Dr. Jesus Tamesis, Dr. José Chan, and other leaders of the Philippine Eye, Ear, Nose, and Throat Society whisked us through customs at the airport and presented us with a printed program of our schedule in Manila. As a result of months of advance planning by our hosts, our stay coincided with the 13th annual meeting of the Philippine Ophthalmological and Otolaryngological Society.

At the banquet of the society, a group of young dancers and performers, who had represented their country at the Brussels Universal and International Exhibition, enchanted us with their spirited folk dances.

On our departure from India, Dr. Y. K. C. Pandit, honorary general secretary of the All-India Ophthalmological Society, wrote, "Your visit has paved a way for the future and has given us a wide range of exchange of thoughts in our own specialty and has created a new atmosphere for a meeting of the East and West."

The overwhelming, generous, warm hospitality from the moment of our arrival until our departure in the various countries of Asia cannot be adequately described in words. In addition to lavish banquets and

entertainment, we received hundreds of acts of thoughtfulness and kindness which were also extended to our wives. To a limited extent we were able to reciprocate by distributing gift packages of eyedrops donated by Alcon, Barnes Hind, Winthrop-Stearns, and the Iso-Sol Companies.

I am sure that we have gained far more than we were able to contribute. We have seen the protean manifestations of tropical eye diseases of which most of us previously had only a textbook knowledge. We made many close friends and developed a tremendous respect and appreciation for our colleagues in Asia.

As we were getting ready to return home, we all felt that we must keep alive and kindle the pleasant relationships we established between Asian and Western ophthalmologists. We also felt that we should encourage others in the medical profession to travel to Asia and become acquainted with some of the grandest and friendliest people in the world.

Much has been written of the role of doctors as diplomats and the role of American doctors abroad as ambassadors of good will.

Dr. Gunnar Gundersen in his presidental address before the 107th annual meeting of the American Medical Association in June, 1958, spoke for all of us when he said, "Physicians here and abroad can do much to advance the cause of freedom and human well being. For medicine, by its very nature, speaks a universal language dedicated to scientific truth, humanitarian service, individual dignity, and world peace."

William John Holmes.

CORRESPONDENCE

COURSE ON INDUSTRIAL OPHTHALMOLOGY

Editor,
American Journal of Ophthalmology:

The incidence of eye injury and the question of work ability of persons with visual impairment, as well as the initiation and management of eye-protection programs, is a problem of major importance in the field of industrial medicine. The training of the industrial physician in ophthalmology and in the management of eye-safety programs has generally been neglected. Though he may be an expert in the properties of various chemicals and their action on the body, though he may be familiar with the effect of trauma on other parts of the body and the proper method of treating injuries of an orthopedic nature, more often than not the problems of eye protection and eye care have not been dealt with in his course of training to any great extent. In an effort to overcome this rather obvious deficiency, The Institute of Industrial Health and the Department of Ophthalmology of the University of Cincinnati are presenting a four-day course entitled "Eyes in industry" on March 9th, 10th, 11th, and 12th at the University of Cincinnati.

Many of the eye problems which arise in industry are somewhat different from the problems seen outside of industry. Questions of administration are not the least of the problems which arise. How shall the eye safety program be instituted and who shall administer it following its initiation in an industry? Shall the company pay for refraction when the wearing of safety glasses is required? What type of emergency facilities should be available for eye care in today's industry? What type of examination is required and how should this examination vary, dependent upon the type of industry and the type of activity which the employee will be doing? What type of activities should be available for people with impaired vision? These are only a few of the many questions which arise in the day-to-day activity of an industrial medical department. It is in an effort to answer these questions that this course is to be presented.

All too often it has been the complaint of the industrial physician that the ophthalmologist is not truly aware of the problems of industry. The pat answer is not necessarily the correct answer and the management of

many of these problems in industry may have to be somewhat different from what would seem to be the case at first glance to the physician outside of industry. The physician specializing in eye problems, the ophthalmologist, might well take a long glance at these problems in order to see whether adequate service is being rendered in and for industry and the industrial employee and whether the present methods in use in industry are actually the best methods.

(Signed) Donald J. Lyle,
Cincinnati, Ohio.

CORRIGENDUM

Editor,

American Journal of Ophthalmology:

In my paper "Angiomatosis retinae . . ." (Am. J. Ophth., **46**:525, 1958) I mentioned that A. B. Reese showed that Coats' disease is a more or less masked telangiectasis of the retinal vessels. Basic credit for this premise should have been given to Herman Elwyn who lucidly described this puzzling disease in the first and second editions (q.v.) of his well-known book *Diseases of the Retina* (New York, Blakiston, edition 2, 1953) chapter 15, page 186, under the heading "Coats' disease: Telangiectasis of retina, . . ." Elwyn confirmed and supported Coats' alternate interpretation that this condition was a primary change in the vessels. Elwyn goes on to say (p. 184): "It is to Coats' credit that he assumed a change in the vessels, especially the smaller ones. These changed small vessels seem to me to conform to our conception of telangiectasis. Therefore, I consider Coats' disease or telangiectasis of the retina to be a disease entity which finds its place among the vascular malformations as outlined in the previous chapter."

(Signed) Derrick Vail,
Chicago, Illinois.

CORNEAL GRAFT TABLE

Editor,

American Journal of Ophthalmology:

Dr. Brendan D. Leahey has informed me by letter that he has been using and publicizing for eight years a corneal graft table similar to the one I described in the November, 1958, issue of THE JOURNAL. I was not aware of his instrument; neither was any one of my colleagues.

(Signed) Rudolf H. Bock,
Palo Alto, California.

BOOK REVIEWS

HIGHLIGHTS OF OPHTHALMOLOGY. By Benjamin F. Boyd. Panama, Republic of Panama, Star and Herald Company, 1958, and New York, U. S. International Medical Book Corporation, 1958, edition 2. 281 pages, illustrated. Price: Not listed.

A few years ago the editor-author, who is professor of ophthalmology, University of Panama School of Medicine and ophthalmologist-in-chief, Seguro Social de Panama, conceived the idea of gathering together significant contributions, mostly from the United States, that were presented at ophthalmic meetings, and condensing them, together with editorial comment, onto mimeographic sheets, like letters. These, in Spanish and English, were sent to subscribers, most of them in the Americas. They met with such a favorable response that the author was compelled to develop this system and to publish them in journal form. The separate journal issues were then re-edited, added to, and finally published in book form in English. The present volume is the second edition, since the first was completely sold out three months after date of publication. This is eloquent testimony to its popular reception.

Part I of the second edition covers the Pan-American Congress of 1957, two of the Chicago Ophthalmological Society meetings of 1957, the 1957 Bedell Lecture (J. Mc-

Lean), and a section on personal interviews. Part II is devoted to advances in ocular surgery and glaucoma. Part III covers the 1957 meeting of the American Academy of Ophthalmology and Otolaryngology, a meeting of ophthalmic surgeons and artificial eye fitters, and a special section on personal interviews.

The success of these volumes is due to the delightfully intimate and informal atmosphere created by the editor, not only in long abstracts of the various papers included, but particularly in his unique and well-executed idea of personal interviews with various authorities on major ophthalmic problems. His questions have a Boswellian and, at times, a Socratic touch. They are skillfully framed to bring out basic ideas. The answers, although properly edited, still reveal the personality and generally sound judgment of those questioned. This meeting of minds is made more intimate by the inclusion of candid photographs of those who were interviewed.

The critical selection of the mass of available material is truly a formidable task. This Dr. Boyd has done very well indeed. His English is fluent and his writing most lucid. The book is strictly clinical in its form and contains valuable material for instant use in our important daily work of taking care of people. All readers are better ophthalmologists because of it.

The printing, paper, and illustrations are excellent and attractive. Unfortunately there is no index, a useful tool in any book of this size. It should be included in subsequent editions. We congratulate Dr. Boyd for his delightful contribution and look forward with pleasure to the regular appearance of more of these "Highlights" (a happy title).

Derrick Vail.

Water and Electrolyte Metabolism in Relation to Age and Sex. Edited by G. E. W. Wolstenholme and C. M. O'Connor. Ciba Foundation Colloquia on Ageing: Volume 4. Boston, Little, Brown and Company, 1958. 327 pages, 85 illustrations, index. Price: $8.50.

An old clinical saw has it that we are as old as our arteries. The reading of this fourth colloquium on ageing may revise this aphorism to "we are as old as our kidneys." In large measure the changes of age are changes in water metabolism which in turn are dependent on alterations in kidney function. Is this then the answer to the question of the usually late onset of glaucoma? This specific question is not answered in this volume but the second paper is a valuable discussion of water balance in the eye by Davson. The remainder of this symposium will be of interest chiefly to physiologists and specialists in renal and ad-renal structures.

David Shoch.

Medical Electrical Equipment. Edited by Robert E. Molloy, M.B. New York, Philosophical Library, 1958. 312 pages, 238 illustrations, index. Price: $15.00.

This small volume covers the whole field of electrical equipment used in the modern hospital from lighting, air-conditioning, and refrigeration to diathermy, motors, and electroencephalography. The text, written by 21 contributors, all connected with British firms, aims to provide authoritative information on the principles, operation, care, and maintenance of medical electrical devices but the treatment is too sketchy and the photographs illustrate only British equipment. The exposition is directed primarily to the nurses and technicians responsible for maintenance rather than to the operators using the equipment.

Hospital administrators will find many useful hints and ideas. More damage is done to the light-fittings of the operating theater by over-cleaning than by years of ordinary use. The multireflector light fittings can be obtained with a space in the center into which can be fitted an automatic color camera capable of taking stills at five- to 20-second intervals. In the personnel paging

system used at St. Thomas Hospital of London, a miniature portable fountain-pen style receiver using transistors is carried in the breast pocket of the staff doctors. On hearing the tone signal, the end containing a telephone ear-piece is placed against the ear for the message.

A similar but more comprehensive book, describing American equipment, with a bibliography and a more adequate index, and written for medical men would be very worth while.

James E. Lebensohn.

So You Have Glaucoma. By Everett R. Veirs. New York and London, Grune and Stratton, Inc., 1958. 64 pages. Price: $2.75.

This small volume is written for the benefit and instruction of the patient with glaucoma and may be recommended for that purpose. Expressed simply in language understandable to any intelligent layman the book gives no encouragement to self-diagnosis or treatment but properly warns of the necessity of adequate ophthalmologic observation and treatment. With the current interest of the public in medical subjects and the psychologic trauma sometimes resulting from the diagnosis of glaucoma, an exposition such as this may be helpful in allaying the fears of the patient and making him more co-operative in pursuing adequate therapy.

William A. Mann.

When-What? The Routine of the Treatment of Amblyopia. (Experiences in the School for Pleoptics and Orthoptics in St. Gallen.) By A. Bangerter and M. Steidele. St. Gallen-Ost, Ehnders, 1958. Price: Swiss f. 6.00.

The booklet consists of two parts. The first part deals with the methods and treatment which vary according to the age of the child, as carried out in Bangerter's "Seeschule." The second part consists of sample forms to be given to the child's parents which explain atropinization, occlusion, and certain forms of home exercises.

The purpose of the first part, which consists of tables in Bangerter's dogmatic and orthodox style, is not quite obvious. It contains mostly catch-words which make sense only to the initiated, and presents nothing that has not already been elaborated in detail in Bangerter's *Amblyopiebehandlung*. Unless these tables are intended for students or orthoptists in Bangerter's institution, as broad outlines for the routine to be followed, this publication seems to be rather superfluous.

Stefan Van Wien.

Indian Year-Book of Ophthalmology. Third Year, 1956.

The largest part of this book, 136 pages, is a collection of abstracts of ophthalmic literature. It also contains an article on the value of plastic lenses implanted into the anterior chamber (eight pages, four figures), a list of the results of examination of candidates for degrees, society proceedings, annual reports of eye hospitals and eye wards for 1955, an outline of ophthalmic relief work, and a list of eye specialists in India.

F. H. Haessler.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

5
DIAGNOSIS AND THERAPY

Pitter, J. and Vihnánek, J. **The treatment of eyes injured with potassium permanganate crystals.** Klin. Monatsbl. f. Augenh. **133**:265-267, 1958.

Vitamin C was directly applied to the cornea and conjunctiva which had been seriously injured by crystals of potassium permanganate in a 13-year-old girl. The improvement and cure were surprisingly fast. They could be repeated in experiments on rabbits. (3 figures, 3 references) Frederick C. Blodi.

Sachsenwenger, R. **The radiologic observation of the optic nerve with water-soluble radio-opaque substances.** Klin. Monatsbl. f. Augenh. **133**:195-202, 1958.

Joduron (1 cc.) in combination with 2 percent procain (1 cc.) is injected retrobulbarly. Tumors of the optic nerve are best outlined by this method. Vascular spasm may follow such an injection and the method should only be applied when the eye has become blind. Two pertinent cases are discussed. (9 figures, 20 references) Frederick C. Blodi.

Schober, Herbert. **Asthenopia when looking at television and its correction.** Klin. Monatsbl. f. Augenh. **133**:254-259, 1958.

Complaints are mainly ascribed to uncorrected hyperopia and heterophoria, especially exophoria. The correction should be given for the far point, but occasionally a +0.50 add may be of advantage. A well-illuminated room is of advantage as the screen occupies only a small part of the visual field. (1 figure, 1 table, 12 references) Frederick C. Blodi.

Swanson, A. A., Rose, H. W. and Taube, J. I. **An effective portable animal platform for Zeiss opton slitlamp examinations.** A.M.A. Arch. Ophth. **59**:465, March, 1958.

The authors describe a platform to hold rabbits for slitlamp examination. (1 figure) G. S. Tyner.

Tóth, Z. **When does the ocular fundus picture not correspond to the blood pressure in the brachial artery?** Klin. Monatsbl. f. Augenh. **133**:237-241, 1958.

The following possibilities can be observed: 1. the blood pressure in the two

brachial arteries is not identical and 2. the fundus picture may not correspond to the systemic blood pressure because of great variations in blood pressure, myocardial damage, antihypertensive treatment, or operation.

Frederick C. Blodi.

Voeroesmarthy, D. and Pénzes, T. **Sphincterotomy by solar coagulation.** Klin. Monatsbl. f. Augenh. **133**:279-281, 1958.

The sphincterotomy was done on an ectopic pupil after a cataract extraction. The sunlight was focused with a 3.5 D lens and regulated with a diaphragm. The exposure time was less than a second. (2 figures, 3 references)

Frederick C. Blodi.

Ziv, Benjamin. **A new contact lens for recording the ERG in rabbits.** A.M.A. Arch. Ophth. **59**:466-468, March, 1958.

The new lens has several advantages; the lids do not close and clear vision is maintained, rotation does not occur, air bubbles are minimized, and a clear cornea is maintained. (4 figures, 5 references)

G. S. Tyner.

6

OCULAR MOTILITY

Alpern, Mathew. **Vergence and accommmodation.** A.M.A. Arch. Ophth. **60**: 355-357, Sept., 1958.

Of the many factors which influence vergence movements, target size has been little considered. Alpern concludes from his experiments that changes in target size can produce changes in vergence, even when accommodation is held fixed. (2 tables, 6 references) G. S. Tyner.

Alpern, Matthew. **Vergence and accommodation.** A.M.A. Arch. Ophth. **60**: 358-360, Sept., 1958.

When an observer suddenly accommodates, the occluded eye moves nasalward. There has been difference of opinion as to whether this associated vergency is related to accommodative effort or to the change in stimulus to accommodation. Alpern's results point to accommodative effort. (1 table, 8 references)

G. S. Tyner.

Girard, L. J. and Neely, R. A. **Agenesis of the medial rectus muscle.** A.M.A. Arch. Ophth. **59**:337-341, March, 1958.

The congenital absence of the medial rectus muscle is reported. The physiologic aspects are discussed. The rotational defect was corrected by transplantation of slips from the vertical muscles. (3 figures, 8 references)

G. S. Tyner.

Holland, Gerhard. **Studies on the influence of the fixation distance and the direction of gaze on horizontal heterophoria (exo- and esophoria).** Arch. f. Ophth. **160**: 144-160, 1958.

In 28 young emmetropes phoria measurements were made with a Maddox rod at distances of fixation varying between 5 meters and 20 centimeters. Recordings were also made with the eyes in elevation and depression and in rotation of 15 and 30 degrees to the right and left. This work confirms the belief that esophoria decreases and exophoria increases as the point of fixation comes closer, that esophoria increases and exophoria decreases when the eyes are depressed, and that adduction tends to increase esophoria, and abduction increases exophoria. (4 figures, 3 tables, 32 references)

Edward U. Murphy.

Kraft, C. and Hoette, E. **Should a squint be operated on before or after the amblyopia has been treated?** Klin. Monatsbl. f. Augenh. **133**:260-265, 1958.

The authors favor a surgical operation before orthoptic or pleioptic treatment.

The main reasons are: 1. the operation may change an anomalous retinal correspondence into a normal one, 2. the Euthyscope can be used more easily when the axes are parallel, 3. preoperative binocular vision is not necessarily followed by postoperative orthophoria, 4. excentric fixation may be improved by the operation and 5. parents and children are perhaps more willing to undergo orthoptic treatment after the initial success of an operation.

Forty-eight patients with excentric fixation were treated in this way and centric fixation was obtained in 33. The result was independent of the age of the patient; the oldest patients in whom treatment was successful were 16, 17 and 20 years of age. (1 figure, 14 references)

Frederick C. Blodi.

Parks, Marshall M. **Abnormal accommodative convergence in squint.** A.M.A. Arch. Ophth. **59**:364-380, March, 1958.

The author studied the accommodation and accommodative-convergence ratio (A:AC) in 1,249 children whose ages ranged from three months to 12 years and who had comitant esotropia or exotropia. He concluded that an abnormal A:AC was responsible for more than 10° difference between the distance and near fixation in half of the children. Regardless of age, bifocals control the symptoms but do not correct the defect in esotropia. D.F.P. almost always normalizes the A:AC while in use in growing children but fails to give correction after being discontinued. In older children there is a tendency to remain normal after D.F.P. is discontinued. Surgery, when justified, permanently improves the A:AC. (10 figures, 11 tables)

G. S. Tyner.

Senita, G. R. and Fisher, E. R. **Progressive dystrophic external ophthalmoplegia following trauma.** A.M.A. Arch. Ophth. **60**:422-426, Sept., 1958.

Two cases are reported of external ophthalmoplegia following trauma. One of the patients was struck on the supraorbital ridge and the other lost consciousness after an explosion. The clinical and pathologic findings are similar to those of progressive dystrophic ophthalmoplegia. (3 figures, 6 references)

G. S. Tyner.

Stanley-Jones, D. **Cybernetics of ocular movement.** Brit. J. Ophth. **42**:595-604, Oct., 1958.

Cybernetics has been defined as the control of machines by machines instead of men. The concept of feed-back is required to understand this and the operation of the thermostat on an electric iron is the simplest example of cybernetic control. Changes in the output (temperature) are made to produce changes in the input (current), this process being called feed-back or refection. The author uses cybernetic analysis to explain nystagmus and oculogyric crises. The pathways of cybernetic impulses are discussed anatomically. Damage to the third nerve nucleus is the probable cause of postencephalitic oculogyric crises. (23 references)

Lawrence L. Garner.

Witzel, S. H. **Congenital paralysis of lateral conjugate gaze.** A.M.A. Arch. Ophth. **59**:463-464, March, 1958.

The unusual occurrence of complete paralysis of horizontal conjugate gaze to both sides in a patient with Klippel-Feil deformity is presented. (5 references)

G. S. Tyner.

7

CONJUNCTIVA, CORNEA, SCLERA

De Mello e Oliveiro, Helion. **Keratitis from an insect bite.** Arq. brasil. de oftal. **21**:21-25, 1958.

A young man was stung in the left eye by an insect; within 48 hours the left eye became red and painful and the vision be-

came impaired. Examination revealed loss of corneal epithelium, striae of Descemet's membrane and an elevated spot which appeared to be the point of entry of the venom. In spite of vigorous therapy, the iris became atrophic, the pupil eccentric and opacities developed in the anterior layers of the crystalline lens. Final corrected visual acuity was 20/60. It is believed that this severe reaction resulted from direct entry of the toxin, acid in nature, into the anterior chamber. The insect was probably a bee or similar insect. (1 photograph, 1 table, 12 references) James W. Brennan.

Foertner, H. G. **The treatment of herpetic corneal diseases with steroids.** Klin. Monatsbl. f. Augenh. **133**:276-278, 1958.

The author has the impression that the topical use of prednisolone is of benefit in superficial and deep herpetic infections of the cornea. (18 references)
Frederick C. Blodi.

Franceschetti, A., Lisch, K. and Klein, D. **Two pairs of identical twins with keratoconus.** Klin. Monatsbl. f. Augenh. **133**:15-30, 1958.

The first pair were 15-year-old girls. One girl had a fully developed keratoconus in each eye. The sister had in one eye only a forme fruste. The other pair were 18-year-old boys. There was a marked keratoconus in all four eyes. (5 figures, 5 tables, 28 references)
Frederick C. Blodi.

Guenther, G. **Keratoplasty in two cases of acute keratoconus.** Klin. Monatsbl. f. Augenh. **133**:40-50, 1958.

Two patients were treated who had an acute aggravation of a pre-existing keratoconus. A perforating corneal transplant was done in both instances. The acute symptoms are caused by extensive rupture in Descemet's membrane. (14 figures, 22 references) Frederick C. Blodi.

Hara, K., Taoka, S. and Kida, S. **Impedance test of the corneal tissue.** Jap. J. Ophth. **2**:160-166, May, 1958.

The most difficult area for ions to permeate is the center of the cornea, which is more permeable in the periphery, and permeability increases in the section where the conjunctiva is present. In the part below the conjunctiva nearly a full permeable condition exists. (4 figures, 1 table, 11 references) Irwin E. Gaynon.

Havener, W. H., Stine, G. T. and Weiss, L. L. **Corneal donor selection by blood type.** A.M.A. Arch. Ophth. **60**:443-447, Sept., 1958.

In 21 corneal transplantations which are reported, the blood types of the donor and recipient are known. The findings are striking. Not one of the four ABO blood-group incompatible transplantations resulted in a clear cornea; 14 of 17 compatible transplanted corneas were clear. (2 tables, 2 references) G. S. Tyner.

Shikano, Shin-ichi. **The Arthus phenomenon in the cornea.** Jap. J. Ophth. **2**: 166-172, May, 1958.

In sensitized rabbits the antigen-antibody reaction in the cornea first caused a marked swelling of the collagen fibers with swelling and decreased pigmentation of the nuclei. Thrombi appeared in the limbal vessels. Leucocytes migrated from the vessels into the parenchyma. New keratocytes appeared to take the place of the swollen cells that had disappeared. (5 figures, 5 references)
Irwin E. Gaynon.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Cleasby, Gilbert W. **Malignant melanoma of the iris.** A.M.A. Arch. Ophth. **60**:403-417, Sept., 1958.

In the last ten years the Stanford Eye

Pathology Laboratory has collected 21 cases of malignant melanoma of the iris. Nineteen have been followed and all are living, with no evidence of recurrence or metasis. The treatment was iridectomy where all of the tumor could be excised. There were 15 enucleations. (19 figures, 1 table, 23 references) G. S. Tyner.

Purtscher, Ernst. **Solid nodules of the iris stroma in mongolism.** Arch. f. Ophthal. **160**:200-215, 1958.

In mongolism there is a syndrome of characteristic stigmata but each child may show only a few of the many possible ones. The author describes in detail the iris nodules which are present in almost all cases and which can be of help in the diagnosis. He examined 23 mongoloid children and 19 showed the typical picture. The development of these nodules was followed from birth in three additional infants and one eye was examined histologically. The nodules were seen to be part of the generalized mesenchymal disturbance typical of the mongoloid state. (7 figures, 2 tables, 34 references) Edward U. Murphy.

Schenk, H. and Papapanos, G. **Injuries to the pigment epithelium of the iris.** Klin. Monatsbl. f. Augenh. **133**:212-217, 1958.

Injuries to the pigment epithelium alone are comparatively rare; four such cases were observed within a period of 18 months. Two patients had an incomplete iridodialysis, one had an incomplete tear, and the fourth patient had a circumscribed defect of the pigment epithelium after a cataract extraction. (5 figures, 8 references) Frederick C. Blodi.

Wadsworth, Joseph A. C. **The uveal tract.** A.M.A. Arch. Ophth. **59**:446-458, March, 1958.

The pertinent literature for the year is reviewed. (125 references) G. S. Tyner.

9

GLAUCOMA AND OCULAR TENSION

Edwards, C. E. and O'Doherty, D. S. **Anti-Parkinson drugs and their relationship to glaucoma.** Georgetown Univ. Med. Center Bull. **12**:15-16, Sept., 1958.

The authors found no reports in the literature which show that the potential danger in using the usual belladonna-like drugs is recognized. The measurement of tension in a random sampling revealed only one minimally abnormal subject; a predisposition to glaucoma is probably of much greater importance. (1 table) Irwin E. Gaynon.

Etienne, R. **Chronic glaucoma and hypertension of the pulmonary artery.** Bull. et mém. Soc. franç. d'opht. **70**:510-517, May, 1957.

The significance of the pressure in the episcleral veins has been recognized in tonographic studies and has been expressed by the classical formula $F = (Po - Pv) \times C$. Physiologic variations in the episceral pressure are rare. Pathologic rise in the episcleral pressure, however, is one of the uncommon causes of glaucoma; inflammatory stenosis of the jugular veins, arteriovenous fistula, mediastinal tumor, hypertension in the region of the pulmonary artery are among its causes. This type of glaucoma is called exogenous glaucoma because the cause of the rise in tension is located outside the eye. Two very interesting case histories are discussed at length with special emphasis on the specific mechanism in each case. (11 references) Alice R. Deutsch.

Friede, Reinhard. **The insertion of hair into the trephine opening in developmental glaucoma.** Klin. Monatsbl. f. Augenh. **133**:94-95, 1958.

Ten to fifty hairs are inserted into the opening. It was successful in one case,

in two others the hair had to be removed.
Frederick C. Blodi.

Hartman, E. and Saraux, H. **Hypervitaminosis A and ocular tension.** Bull. et mém. Soc. franç. d'opht. **70**:446-456, May, 1957.

The syndrome "acute hydrocephalus of infants" has been described by Julien Marie and Germain Sée in 1951. Pronounced bulging of the fontanelles, vomiting and somnolence are considered to be the essential signs and symptoms, and they are ascribed to an increase of intracranial pressure following over-dosage of natural vitamin A in oil. In adults similar but less spectacular increases in intracranial pressure were found also only after the ingestion of natural vitamin A in oil. After intramuscular injection or after the oral use of a synthetic vitamin A, complications of this kind were never noted. The ocular tension in hypervitaminosis A has never been checked routinely. Animal experiments were made on rabbits. A considerable reduction in tension was found after 48 hours, again only if natural vitamin A was given by mouth. Retrobulbar and intramuscular injections were ineffective.

In the clinical investigation 25 patients were studied. They took 300,000 units vitamin A in oil as a daily dose. In 19 patients considerable hypotony occurred, starting 48 hours after medication. This hypotony persisted during the period of sustained medication. In five cases of chronic glaucoma and one of uveitis no variations in tension occurred. The hypotensive action of vitamin A is ascribed to an increased elimination of vitamin C in the urine and a corresponding decrease of ascorbic acid in the aqueous. (5 figures, 1 table, 3 references) Alice R. Deutsch.

Javid, Manucher. **Urea—new use of an old agent: reduction of intracranial and intraocular pressure.** Surg. Cl. North America, pp. 907-928, Aug., 1958.

Urea is effective in reducing intraocular pressure and a 30-percent solution is suggested in preparation of a patient for surgery for acute glaucoma.
Irwin E. Gaynon.

Jones, R. F. **Glaucoma following radiotherapy.** Brit. J. Ophth. **42**:636-638, Oct., 1958.

A case of glaucoma is described in which radiotherapy is thought to be the etiologic agent. Histologic study is not available, but the finding of tortuous, dilated newly-formed vessels in the iris and chamber angle after therapy is significant. Unfortunately tonography, a means of distinguishing between an impaired outflow and hypersecretion, could not be done. (6 references)
Lawrence L. Garner.

Küchle, H. J. and Rohrschneider, W. **Electroshock and intraocular pressure. IV. The role of the midbrain in changes of intraocular pressure.** Arch. f. Ophth. **160**:186-199, 1958.

Comparative studies on rabbit eyes and human eyes with the use of electroshock show changes in intraocular pressure in the same direction and lead the authors to state that the results of such animal experimentation may be applicable to man. (3 figures, 5 tables, 36 references)
Edward U. Murphy.

Legrand, J., Hervouet, F. and Lenoir, A. **The pathologic anatomy of iridencleisis. Reasons for success and failure.** Bull et mém. Soc. franç. d'opht. **70**:457-483, May, 1957.

The authors discuss the pathologic anatomy of the two main varieties of iridencleisis, the Sourdille-Pillat and the classical Weekers techniques. It took ten years to collect the necessary material, namely a sufficient number of eyes with

well filtering and nonfiltering iridencleisis scars at the time of enucleation without secondary changes not related to the surgery which had been done. Adequate numbers of serial sections were made.

Clinically, iridencleisis scars appeared in four distinct forms: flat, more or less pigmented scars; prominent, hyperemic fibrous scars; mildly prominent occasionally pigmented scars; and bullous, transparent, soft scars. Only eyes with a bullous scar had either normal or subnormal tension. The normalization of tension was ascribed to two factors: 1. to a mechanical process through fistulization along an iris incarcerated in a scleral wound with disengagement of the chamber angle and 2. an indirect traumatic neurovascular process acting on the production and elimination of the aqueous. In spite of the fact that iridencleisis has been considered to be easy in technique, even mild deviations from the basic specific design always has caused difficulties which result in serious complications. An exact vertical incision at the surgical limbus is a definite requirement. (9 figures, 13 references) Alice R. Deutsch.

Petersen, Hans Peter. **Exfoliation of the anterior lens capsule with glaucoma.** Acta ophth. **36**:375-380, 1958.

Exfoliation of the lens capsule can occur with or without glaucoma; 84 patients with exfoliation were observed for a period of one to 10 years. The difference between an eye which has exfoliation with glaucoma and one without it is in the pigment distribution in the anterior chamber angle. Statistically speaking, glaucoma was more prevalent in eyes with the heavier pigment deposit in the angle. It is doubtful, however, whether the pigmentation is secondary or primary to the glaucoma; the two may develop together. Also, the pigment scattering is not the only factor in capsulary glaucoma. (17 references) John J. Stern.

Plamondon, Marc. **Physiology of primary glaucoma (a new pathogenic concept).** Laval Med. **26**:12-27, June, 1958.

Whether primary glaucoma is caused by a mechanical obstruction of the outflow channels or by a vascular abnormality is still much discussed. The author feels that both points of view are true and presents a theory which combines and unifies these apparently divergent concepts. The iris is as important as the ciliary body in the formation of aqueous humor and when there is hyposecretion from the iris for vascular reasons, a disequilibrium is set up and the iris-lens diaphragm moves forward. In acute glaucoma an arteriolar spasm in the iris causes anoxia of the sphincter and thus mydriasis. Anoxia also causes an edema of the root of the iris which blocks the angle. (26 references) Edward U. Murphy.

Porter, G. LeRoy. **Incidence and detection of glaucoma by routine tonometry.** Tr. Am. Acad. Ophth. **62**:54-62, Jan.-Feb., 1958.

Detailed studies were made in all patients having a tension of 25 mm. Hg (Schiøtz) in a series of 2,000 patients over the age of 40 years. Patients with known glaucoma, or those who might be expected to have altered tension were excluded. Fifty-two individuals (2.6 percent) were found to have a pressure over 25 in at least one eye. Of this number, 35 were found to have glaucoma; in 16 there were field and fundus changes and in the 19 others the water drinking test was positive or the tension was repeatedly over 32 mm. The 17 other patients were not found to have glaucoma by these tests. However, an additional nine cases of glaucoma were found in patients whose initial tonometric reading had been below 25 mm. Consequently, in 2,000 patients, 44 were found to have glaucoma, 35 of them on the basis of a screening value of

25 mm. Hg on initial tonometry, and nine of them not found by this criterion.

Various tests for glaucoma and the results of a questionnaire are discussed. The most interesting finding from the latter is that one-third of Board certified ophthalmologists stated they did not use routine tonometry. There is a discussion following the article. (3 tables, 15 references)

Harry Horwich.

Sampaolesi, R. **Tonography with the Schiøtz tonometer and Merté's special support.** Arch. oftal. Buenos Aires **33**:101-104, April, 1958.

Tonography with the Schiøtz tonometer seems to have been first used as a standard procedure by König (König, H. Klin. Monatsbl. f. Augenh. 126:401-409, 1955). who calculated the facility and rate of aqueous flow by means of special tables. In order to overcome the difficulty encountered in resting steadily the instrument upon the patient's cornea, Merté conceived a supporting device consisting of a light metallic shaft which holds the tonometer firmly in position and is fastened to the subject's head with a rubber band. A plastic lid retractor is used which is claimed to exert no undue pressure on the eyeball.

Comparative recordings made in 10 patients with both this technique and Mueller's electronic tonometer were in close agreement. It must be remembered, however, that because of inadequate freedom of the footplate and plunger assembly from the collar, the former is prevented from following freely the slight displacements which the cornea displays during the experiment; with the electronic tonometer, on the other hand, the lateral wobble has no appreciable effect on the current generated. (2 figures, 1 graph, 7 references) A. Urrets-Zavalia, Jr.

Schrader, K. E. and Medler, A. **Etiology and therapy of the glaucoma secondary to central vein thrombosis.** Klin. Monatsbl. f. Augenh. **133**:189-194, 1958.

During the last six years 45 patients with central vein occlusion were seen and 29 of them developed a secondary glaucoma. The patients who developed glaucoma were all more than 50 years old and most of them had a generalized arteriosclerosis. Iridencleisis is the operation of choice if the eye is not totally blind. The operation was done on 12 eyes and only one had to be enucleated later. (2 figures, 11 references) Frederick C. Blodi.

Törnquist, Ragnar. **Angle-closure glaucoma in an eye with a plateau type of iris.** Acta ophth. **36**:419-423, 1958.

Acute glaucoma in a patient with a normal axial chamber depth but a narrow angle (plateau iris) is described. The attacks occurred during close work but not when the pupil was enlarged in darkness or by mydriatics. (1 figure, 17 references)

John J. Stern.

Trantas, N. G. **The red ring of the chamber angle and the miotics.** Bull. et mém. Soc. franç. d'opht. **70**:484-509, May, 1957.

The red ring of the iridocorneal angle which is visible in gonioscopy corresponds to the blood band in Schlemm's canal. Its presence (positive test) or absence during gonioscopic examination has been considered to be a valuable diagnostic sign and an important aid in the evaluation of the stage of the glaucoma. It also is helpful in the appraisal of the action of the miotics. The red ring was visible in 91 out of 100 normal eyes, but only in 84.84 percent of the normal eyes of patients with unilateral chronic glaucoma. An even lower percentage of positive tests was found when the second eye was considered to be borderline. According to the progress of the glaucomatous process the visibility of the blood in Schlemm's canal decreased, probably in

proportion to disturbed relations with the general circulation, the circulation of the brain and the cerebrospinal fluid. In early chronic glaucoma the test was positive in 44 percent and increased to 66 percent with the use of pilocarpine. In more advanced cases a 16.8 percent of positive tests could be increased to 60 percent but had to be supported eventually by the use of eserine and Diamox, demonstrating the increasing difficulty of medical control.

The significance and usefulness of this test is explained; case histories are quoted with special emphasis on the significance of the test in individual patients. The size and the weight of the contact lens was considered to be irrelevant. The test is recommended for diagnosis and medical control of glaucoma.

Alice R. Deutsch.

Wolter, J. R. and Lubeck, M. J. **Glaucoma secondary to occlusion of the central retinal artery.** Klin. Monatsbl. f. Augenh. **133**:179-189, 1958.

A 70-year-old man developed glaucoma about two months after arterial occlusion. On histologic examination the angle was found to be open. Small, new-formed vessels were seen on the iris and over the posterior part of the trabecular meshwork. There was also a membrane with vessels on the inner surface of the papilla. Part of the retina remained viable because it was supplied by a ciliary artery. Two short posterior ciliary arteries were also occluded. (10 figures, 10 references)

Frederick C. Blodi.

10

CRYSTALLINE LENS

Colding, A. and Edmund, C. **General anesthesia in 104 cataract operations.** Acta ophth. **36**:407-415, 1958.

General anesthesia in 104 cataract operations consisted of a combination of pentothal sodium, succinyl choline, N_2O-O_2 and Largactil. After comparing the results with those of 45 operations under local analgesia, the authors feel that the operation can be done just as safely under general as under local anesthesia, if a special technique is used. In nervous and apprehensive patients and in cases of complicated cataract the results were even better under general anesthesia. (7 tables, 6 references)

John J. Stern.

Csillag, F. **Puncture of the capsule before a cataract extraction.** Klin. Monatsbl. f. Augenh. **133**:66-71, 1958.

When it is difficult to grasp the lens capsule with a forceps (intumescent or hypermature cataract), a preceding puncture of the capsule is advised. The puncture is done in the upper third of the lens with a thin needle. The capsule is grasped with a forceps at the lower third and extracted by tumbling. One hundred cases were operated on in this way and 24 extractions were truly intra-capsular. (13 references)

Frederick C. Blodi.

Fechner, P. Ullrich. **Cataract extraction by expression.** Klin. Monatsbl. f. Augenh. **133**:71-82, 1958.

In this report on a trip to India the method of Dr. Mathra Das is described. The section is corneal, iridectomy total, and delivery by expression only. Das has performed more than 225,000 cataract extractions by this method. The duration of the entire operation is one minute and only 3 percent of the operations are extracapsular. Loss of vitreous occurred in about 3 percent of the cases, even though he uses no akinesia and no sedation. Secondary glaucoma is a frequent postoperative complication after using this method. (8 figures, 41 references)

Frederick C. Blodi.

Friede, Reinhard. **Technique of synecholysis in complicated cataracts.** Klin.

Monatsbl. f. Augenh. **132**:884-885, 1958.

A new synechotome is described. The spatula forms an angle of nearly 90 degrees with the handle. (1 figure)

Frederick C. Blodi.

Gasteiger, H. **The implantation of anterior chamber lenses.** Klin. Monatsbl. f. Augenh. **132**:609-617, 1958.

Of 20 aphakic patients five see 6/9 or better without any additional glass and five others have this vision with a small correction. Three of the patients saw definitely worse after the second operation in which the lens was implanted. One had a purulent infection with loss of the eye, in one patient the second operation was followed by an epithelial downgrowth with loss of vision and in the third patient a tilting of the lens prevented good vision. In another four patients a secondary or inflammatory membrane reduced vision below 6/24. The method is advised only for young patients with unilateral cataracts. (1 table, 11 references)

Frederick C. Blodi.

Gormaz B, Alberto. **Corneal "flap" incision for cataract operation.** Brit. J. Ophth. **42**:486-493, Aug., 1958.

A new type of cataract incision based upon the halving procedure of Wheeler is described. In this instance the superficial portion of the corneal incision does not coincide with the deeper incision. Preparation is in the usual manner: a flap of conjunctiva is not used and a Gill knife is used to make the grooved incision just within the cornea at the limbus slightly beyond the 3- and 9-o'clock positions. The Gill knife is then used to split the cornea about 1.5 mm. along this groove. A keratome may then be used to enter the chamber and scissors are used for enlargement of the section. A preplaced suture is applied in the 12-o'clock position before removal of the lens, and three sutures are applied post-placed on each side of this central suture upon closure. It is suggested that this type of corneal wound, if properly sutured, develops greater tensile strength. The procedure has been used in more than 50 cases. (8 figures, 29 references)

Lawrence L. Garner.

Hartman, E., Saraux, H. and Haye, C. **Cataract extraction after direct disinsertion of the zonule.** Ann. d'ocul. **191**:397-410, June, 1958.

The authors report 138 cataract extractions performed by the Kirby technique of direct zonular rupture. Of these 34 were considered pathologic and the rest senile. In the group of 104 senile cataracts there were 16 (15 percent) capsular ruptures. There was a 5 percent incidence of hyphema. In the pathologic group the incidence of capsular rupture was 24 percent. The only loss of vitreous occurred in this group. The authors feel that this technique is the safest of all for cataract extraction. (6 figures, 14 references)

David Shoch.

Hartstein, Jack. **Studies of the crystalline lens in humans receiving galactose over prolonged periods.** A.M.A. Arch. Ophth. **59**:406, March, 1958.

The author points out that none of the patients receiving galactose developed cataracts. (9 references)

G. S. Tyner.

Jaffe, N. S. and Light, D. S. **Surgery of developmental cataract.** A.M.A. Arch. Ophth. **59**:407-416, March, 1958.

The authors support the current belief that discission of developmental cataracts should be abandoned in favor of linear extraction. They describe their technique which has been used on 17 eyes. They make a limbal incision, 6 to 8 mm. long, which is closed later with four 6-0 chromic sutures. A peripheral iridectomy is followed by capsulectomy. The soft lens material is removed by means of a

spoon or hook and the anterior chamber is irrigated. Air is injected into the anterior chamber after closure. (5 figures, 19 references) G. S. Tyner.

Krakau, C. E. T. **Experiences with "Flieringa's Ring" in surgery for luxated lenses.** Acta ophth. **36**:416-418, 1958.

Flieringa in 1953 described a method facilitating the extraction of luxated lenses. A stainless steel ring is sutured to the eye around the cornea which stabilizes the bulb after it has been opened. The method was used in luxation of the lens into the vitreous in six cases and, into the anterior chamber in one. No loss of vitreous was experienced, and the method is recommended for other intraocular surgery where a loss of vitreous may be anticipated. (1 figure, 1 table, 1 reference) John J. Stern.

Krueger, K. E. and Jaehner, H. **Bilateral aphakia and binocular vision.** Klin. Monatsbl. f. Augenh. **132**:720-726, 1958.

Of 66 patients 63 had simultaneous perception, and 60 had fusion with amplitudes. 53 patients had depth perception. (3 tables, 4 references) Frederick C. Blodi.

Malbrán, Enrique. **Enzymatic zonulolysis in cataract surgery: a preliminary report.** Arch. oftal. Buenos Aires **33**:113-114, May, 1958.

If irrigation of both the anterior and the posterior chamber with a 1/5000 solution of alpha-chymotrypsin—a proteolytic and milk-curdling enzyme of the pancreatic secretion—in physiologic saline is effected after completion of the limbal incision, the substance of the zonular fibers is readily dissolved; removal of the lens may then be performed with considerable ease and with little risk of rupture of the capsule (Barraquer).

Seven eyes with cataractous lenses, in six patients whose ages were 40, 45, 59, 65, 65, and 67 years, were operated on with this procedure; the erisophake was used in five of them, and Castroviejo's foreceps in the remaining two. In all of them an intracapsular extraction could be carried out, with results which do not differ significantly from those obtained by most surgeons in such a short series of cases. The zonular resistance was found to be definitely lowered in six eyes. No immediate postoperative complication was noted; the follow-up period was apparently of only a few weeks. (1 reference) A. Urrets-Zavalia, Jr.

Mamo, J. G. and Leinfelder, P. J. **Growth of lens epithelium in culture. I. Characteristics of growth.** A.M.A. Arch. Ophth. **59**:417-419, March, 1958.

The authors have successfully grown human lens epithelium in roller tube cultures. Cells survive up to 21 days. (2 figures, 4 references) G. S. Tyner.

Mamo, J. G. and Leinfelder, P. J. **Growth of lens epithelium in culture. II. Effects of oxygen tension.** A.M.A. Arch. Ophth. **59**:420-422, March, 1958.

In a second article the authors show that lens epithelium grows best under aerobic conditions. (3 figures, 6 references) G. S. Tyner.

Pau, H. **The time factor in expulsive hemorrhages.** Klin. Monatsbl. f. Augenh. **132**:865-869, 1958.

Six such hemorrhages were observed among 1,520 cataract extractions and 53 cases from the literature are reviewed. Less than a third of the hemorrhages occurred during the operation, another third three to six hours after the operation and the others later (up to nine days postoperatively). Usually the hemorrhage is not the result of mere spontaneous bursting of a vessel. More often there are other factors, such as a sudden increase of pressure during coughing or sneezing,

myopia, glaucoma or atherosclerosis. (1 figure, 1 table, 25 references)

Frederick C. Blodi.

Picó, Guillermo. **The management of endophthalmitis following cataract extraction.** A.M.A. Arch. Ophth. **59**:381-385, March, 1958.

The author states that either chloramphenicol or tetracycline is the drug of choice in this type of infection. The concurrent use of steroids is indicated to prevent damage from inflammation. Five cases are reported. (1 table, 18 references) G. S. Tyner.

Pinkerton, Ogden, D. **Cataract associated with congenital ichthyosis.** A.M.A. Arch. Ophth. **60**:393-396, Sept., 1958.

Two rare cases are presented in Japanese male siblings in whom cataract was associated with congenital ichthyosis. Whether the changes were coincidental is not known. (9 figures, 4 references) G. S. Tyner.

Richards, R. D. and Ellis, P. P. **Subluxation of the lens following detachment surgery.** A.M.A. Arch. Ophth. **60**:472-474, Sept., 1958.

The authors report a case of a subluxation of the lens one year after retinal detachment surgery, apparently due to traction bands. (2 figures, 4 references) G. S. Tyner.

Ryan, Edward D. M. **The surgery of cataract in megalocornea.** A.M.A. Arch. Ophth. **59**:386-388, March, 1958.

The author reports successful cataract extraction in both eyes of a patient with megalocornea. Although both lenses were subluxated, extraction was accomplished with an erisophake without loss of vitreous. (4 references) G. S. Tyner.

Schillinger, R. J., Shearer, R. V. and Levy, O. R. **Animal experiments with a new type of intraocular acrylic lens.** A.M.A. Arch. Ophth. **59**:423-434, March, 1958.

A lens shaped like a collar button and made from methacrylate is inserted into the pupil to straddle the iris. Successful implantation was accomplished in only one of 13 animal eyes. The formation of pupillary membranes and corneal opacification complicated the procedure. (17 figures, 7 references) G. S. Tyner.

Sivasubramaniam, P. **Unusual traumatic deformity of the lens.** Brit. J. Ophth. **42**:634-635, Oct., 1958.

A bizarre lens injury resulted from contusion of the eye of a 12-year-old boy with laceration and prolapse of the iris. Biomicroscopy revealed a "buckling" of the posterior lens surface without actually producing a tear in the lens capsule. In two cases previously described the fold was ascribed to a partial rupture of the zonule. Vision is reduced. (3 figures, 1 reference) Lawrence L. Garner.

11

RETINA AND VITREOUS

Alberth, B., Balint, A. and Kosa, A. **Audiometric determinations on patients with pigmentary degenerations of the retina.** Klin. Monatsbl. f. Augenh. **132**: 797-806, 1958.

Fifteen patients with nonhereditary retinitis pigmentosa were examined. All had a somewhat reduced hearing, especially in the higher frequencies and around C5. Hearing is not lost at the same rate as the central vision, but there is frequently a correlation with the loss of the visual field. Ten patients were treated with vitamins and implantation of placenta; five of them showed improvement. (4 figures, 38 references)

Frederick C. Blodi.

Arrechea, A., Oyenard, A. H. and Reca, O. **Fundus lesions in polycythemia.** Arch. oftal. Buenos Aires **33**:91-94, April, 1958.

Whenever a severe excess in the number of circulating red corpuscles exists, be it primary (as in Vaquez-Osler syndrome) or only secondary to other morbid entities (such as congenital heart disease, pulmonary sclerosis and emphysema), the clinical picture of cyanosis retinae is seen. It is characterized by dilation and abnormal tortuosity of the veins, which are deep purple in color, an overall dark red discoloration of the fundus; congestion and edema of the disc, and scattered hemorrhages.

Two cases are reported in which the condition appeared as a result of old fibrotic pulmonary lesions with cardiac insufficiency. In one, that of a 32-year-old man, the red blood cell count was of 7,200,000 per cu. mm., while in the other, that of a 34-year-old woman, it was of 7,800,000; hemoglobin concentration was of 26.72 and 24.48 g., respectively. (21 references) A. Urrets-Zavalia, Jr.

Berens, Conrad. **Special pins and forceps for retinal detachment surgery.** A.M.A. Arch. Ophth. **60**:498, Sept., 1958.

Berens has devised new flatter-rounded pins and special forceps to minimize premature displacement of the pins. He recommends many small draining areas rather than one or two large perforations of the sclera. (1 figure, 2 references)

G. S. Tyner.

Boberg-Ans, J. **Retinoblastoma and its treatment.** Acta ophth. **36**:475-482, 1958.

Radio-active cobalt discs after Stallard were used in three eyes with retinoblastoma in two children. All three eyes were saved. (7 figures, 8 references)

John J. Stern.

Duke, J. R. **Pseudoglioma in children: aspects of clinical and pathologic diagnosis.** South. M. J. **51**:754-759, June, 1958.

Forty eyes enucleated from infants were studied pathologically, and classified according to Sanders. Not all were suspected of retinoblastoma, but might not have been removed were not the possibility present. Type 1, persistent hyperplastic primary vitreous, occurred once in a child four weeks old. The anterior chamber was flat, the iris was atrophic, and a cataract was forming Retinoblastoma occurs in a normal-sized eye with a normal anterior chamber, with no visible ciliary processes, and without cataract. Type 2, organized vitreous mass, occurred 15 times. In this series six were due to nematode infestation, two were vitreous hemorrhage, one was a purulent abscess, and six were metastatic infection from a febrile illness. Of Type 3 chorioretinitis, only two cases occurred. One was probably toxoplasmosis. Type 4, exudative retinitis, occurred 18 times in patients from eight months to eight years of age. Two-thirds were males, and in half of the cases symptoms were of three months duration or less. There was detachment of the retina, pink staining albuminous material in the subretinal space, retinal edema with bladder cells and no involvement of the uveal tract. Type 5, massive retinal fibrosis, was represented by only case which might have been a variant of Type 2. Type 6, retrolental fibroplasia, may be disappearing. Of three cases, one showed a congenital retinal fold, but none were thought to be retinoblastoma.

There were no cases of Type 7, other tumors. (3 tables, 11 references)

Paul W. Miles.

Gottfredsen, E. and Krogsgaard, A. R. **Reversibility of hypertensive fundus changes during drug treatment.** Acta ophth. **36**:540-545, 1958.

Forty-four patients with severe hypertension and hypertensive retinopathy of third and fourth degree were treated with ganglion-blocking and centrally acting drugs. Choked disc, fresh exudates, and

hemorrhages subsided in most cases; vascular changes, except angiospastic ones were influenced but little. Choked disc is the first sign to disappear, (two and one-half months), followed by exudates and hemorrhages (four to five and one-half months) and hyaline plaques (up to two years). In a few patients the fundus changes improve even though the blood pressure is not lowered as much as desired. (3 figures) John J. Stern.

Gregory, Irene, D. R. **Familial macular defects.** Brit. J. Ophth. **42**:617-619, Oct., 1958.

Four members of one family presented macular changes in one or both eyes strongly suggestive of Best's disease. The lesions were atrophic in most cases and when cystic were considered to be a phase of Best's disease. At times the lesion looks exudative but in all cases the macular and paramacular region is involved. Eye lesions in this condition remain stationary and frequently fairly good central vision is retained. (5 figures, 1 reference)

Lawrence L. Garner.

Hallgren, Bertil. **Retinitis pigmentosa in combination with congenital deafness and vestibulocerebellar ataxia; with psychiatric abnormality in some cases. A clinical and genetic study.** Acta Genet. & Stat. Med. **8**:97-104, 1958.

A preliminary report is made of a clinical and genetic investigation of the rare syndrome retinitis pigmentosa, congenital deafness and spinocerebellar ataxia, with psychiatric disorder in some cases. The series consists of 88 cases in 49 families.

All affected individuals who had reached the age of 10 and had been examined by the author, had both retinitis pigmentosa and congenital deafness. Cataract occurred in 11 of the patients with retinitis pigmentosa. All affected individuals about whom information was available showed disturbances of gait of a vestibulocerebellar type, 30 percent were mentally defective and 9 percent had a psychosis.

The genetic analysis plainly indicates that the disorder is due to a single recessive gene mutation which manifests itself in retinitis pigmentosa as well as in congenital deafness. There also seems to be a relationship between these two conditions and mental deficiency and psychosis. (2 tables, 10 references)

Author's summary.

Heaton, J. M., McCormick, A. J. A. and Freeman, A. G. **Tobacco amblyopia; clinical manifestation of vitamin B_{12} deficiency.** Lancet **2**:286-290, Aug. 9, 1958.

The authors present extensive evidence that the retina and optic nerve are unduly sensitive to tobacco in persons with even mild vitamin B_{12} deficiency.

Irwin E. Gaynon.

Hughes, Wendell L. **Overlapping technique to produce a scleral fold.** A.M.A. Arch. Ophth. **60**:502-504, Sept., 1958.

Hughes uses the halving technique of Wheeler to produce a scleral fold and to more securely hold the area because of the increased area of the raw surfaces which must grow together. The method can be combined with the polyethylene tubing technique. (3 figures)

G. S. Tyner.

Jochmus, H. **Follow-up examinations in retrolental fibroplasia.** Klin. Monatsbl. f. Augenh. **133**:249-253, 1958.

Eleven children were examined, ten of whom had been prematurely born. In eight children the final, full-blown stage was present. In a pair of fraternal twins the heavier child was more severely affected. (34 references)

Frederick C. Blodi.

Kojima, Y. **Capillary fragility in eye diseases, with special reference to central**

retinitis. Acta Soc. Ophth. Japan. **62**: 1074-1079, Aug., 1958.

The fragility of the skin capillaries is studied in various ocular conditions. An increase in the fragility is observed in sclerosis, glaucoma and central retinitis. Kojima believes that there is a causal relationship between the increased fragility of the capillaries and the glaucoma and central retinitis. (9 tables, 23 references)

Yukihiko Mitsui.

Mark, T. and Vit, H. **Fundus changes in uremia.** Wiener med. Wchnschr. **108**: 461-462, May 24, 1958.

The fundus changes in 100 unselected patients who died in uremia are described and summarized in tabular form; 30 showed only changes compatible with advanced age, 29 had angiospastic retinopathy, and 25 showed vascular sclerosis. (6 tables) Edward U. Murphy.

Meyer-Schwickerath, G. and Helferich, E. **Treatment of retinoblastoma.** Klin. Monatsbl. f. Augenh. **132**:806-817, 1958.

Seventeen eyes with retinoblastoma were treated by light coagulation; the lesion was unilateral in five and the eyes were later enucleated. The area around the tumor is first coagulated and then the tumor itself is treated intensively. Among the 12 bilateral lesions nine appear cured. Repeated coagulations may be necessary. (5 figures, 10 references)

Frederick C. Blodi.

Paufique, M. L. and Royer, J. **Ocular manifestations in the macroglobulinemias.** J. Med. Lyon **39**:525-530, June 20, 1958.

In three affections abnormal circulating proteins constitute the essential part of the syndrome. These are: essential macroglobulinemia of Waldenstrom, hyperglobulinic purpura of Waldenstrom, and essential cryoglobulinemia. The increased viscosity of the blood gives rise to the fundus picture of enlarged, irregular, tortuous veins and retinal hemorrhages. In such cases the conjunctival circulation may be observed to be retarded markedly or to be stopped completely after the application of ice to the closed lids. (2 figures, 84 references)

Edward U. Murphy.

Peckham, R. H. and Hart, W. M. **Critical flicker frequency, photo-chemical mechanisms, and perceptual responses.** A.M.A. Arch. Ophth. **60**:461-471, Sept., 1958.

This article on flicker fusion is designed to refine the use of this method in tests of retinal function. (3 tables, 3 figures, 65 references) G. S. Tyner.

Rosengren, B. and Törnquist, R. **Scleral indentation by means of plastic plugs in surgery of retinal detachment.** Acta ophth. **36**:426-431, 1958.

A method is described in which a plastic plug is used to create an indentation over the site of a retinal tear in detachment of the retina. By assuring contact between the indented sclera and the retina the time of rest in bed can be shortened. Combined pressure by a plug from the outside and air in the vitreous from within, seems to facilitate healing in some cases. The method, first proposed by Custodis, has been used in 62 cases; in 47, or 76 percent, it was successful. (4 figures, 6 references)

John J. Stern.

Sedan, Jean. **Seven evaluations of retinal detachments catalogued as traumatic.** Ann. d'ocul. **191**:358-362, May, 1958.

The author reviews seven cases in which trauma was alleged to play a part in the etiology of retinal detachment. In each case disability claims were allowed by the judiciary on this basis. The author feels that trauma is a very rare cause of retinal detachment and shows that in the cases cited the trauma frequently preceded the retinal detachment by months

or years and that there were almost always other organic changes such as high myopia, peripheral choroiditis, or macular degeneration. (1 table, 11 references)

David Shoch.

Seitz, R. **The so-called dishoric or capillarosis spots of the retina.** Klin. Monatsbl. f. Augenh. **133**:87-93, 1958.

These spots are small, whitish-yellow, sharply outlined and lie near small vessels. They occur in patients with cardiovascular hypertension. One such eye could be sectioned and these spots were found to correspond to cyst-like spaces in the nerve-fiber layer. An acute swelling of ganglion cells probably precedes the cyst formation. (6 figures, 20 references)

Frederick C. Blodi.

Siegert, P. **Temporary (fleeting) detachment in preëclampsia.** Klin. Monatsbl. f. Augenh. **132**:727-731, 1958.

In two patients accelerated delivery cured the detachment rapidly. (10 references)

Frederick C. Blodi.

Vannas, M. **Strengthened suturing in ablation and myopia.** Acta ophth. **36**:432-434, 1958.

Vannas comments on a motion picture showing a modified scleral buckling operation for detachment. (2 figures)

John J. Stern.

Vannas, S. and Orma, H. **The treatment of arteriosclerotic chorioretinopathy.** Acta ophth. **36**:601-612, 1958.

Seventy-one patients with impaired vision, field changes and sclerotic fundus changes were divided into four groups. Improvement of vision by at least two lines or complete disappearance of metamorphopsia were considered as positive therapeutic results. Two-thirds of the group receiving alternating heparin and vitamins A and E showed positive results (100 mg. heparin intravenously daily for two to four weeks, then twice weekly for two to six months; 30,000 units vitamin A, 70 mg. vitamin E, 1 tablet four times daily for four months to three years). One-third of the patients receiving vitamin A and E alone, or Hydergin (Sandoz) alone, improved; and not one in the group receiving a placebo showed an improvement. (8 figures, 3 tables, 22 references)

John J. Stern.

Wagener, H. P. **Some recent studies on Eales' disease.** Am. J. M. Sc. **236**:250-263, Aug., 1958.

Wagener summarizes recent publications on this subject.

12

OPTIC NERVE AND CHIASM

Badtke, G. **The histology and etiology of atypical colobomas of the fundus and the disc.** Klin. Monatsbl. f. Augenh. **132**: 626-634, 1958.

Histologic examination of the microphthalmic eye of an eight-months-old child is described. The coloboma extended nasally and upward in the fundus. In the area of the coloboma the pigment epithelium and the choroid were missing. The retina showed glial degeneration. It is therefore a typical coloboma, but in an atypical direction. It is possible that a faulty closure of an atypically situated fetal cleft may produce such colobomas. (9 figures, 1 reference)

Frederick C. Blodi.

D'Arrigo, Pasquale. **Clinical and therapeutic considerations of neuromyelitis optica.** Arch. di ottal. **62**:93-114, March-April, 1958.

This article reviews the literature and reports two cases of optic neuritis associated with paraplegia and transverse myelitis. The typical case begins with brief intervals of blurred vision and loss of field, fever, occasional rash, psychic

disturbance, blurring of disc margins and papilledema followed in some cases by atrophy, flaccid paralysis, brief hemifacial pain, increased albumin content and mild lymphocytosis of cerebrospinal fluid, and impotence. It is fatal in half of the patients.

In recent years, the etiology has been doubtful. One must rule out multiple sclerosis, acute disseminated encephalomyelitis, diffuse cerebral sclerosis, and Devic's disease.

The cases reported occurred in patients aged 31 and 22 years. After large doses of penicillin, streptomycin, and aureomycin, the symptoms improved. Vision improved from 1/20 to 2/10, and from 1/50 to 1/20 in the first patient. The second patient had permanent total loss of vision in the right eye. (96 references)

Paul W. Miles.

Dodge, H. W., Jr., Love, J. G., Craig, W. M., Dockerty, M. B., Kearns, T. P., Holman, C. B. and Hayles, L. B. **Gliomas of the optic nerve.** A.M.A. Arch. Neurol. & Psychiat. **79**:607-621, June, 1958.

The authors review 46 pathologically verified cases of primary glioma of the optic nerve, chiasm, or tract, which were observed during 41 years at the Mayo Clinic. Twelve of the lesions were limited to the optic nerve and 34 were in the chiasm or the optic tract. Since 33 were found in children it is probable that these tumors have a congenital origin.

The diagnostic signs and symptoms are discussed. Proptosis was the outstanding findings in patients who had a lesion of the optic nerve; bilateral loss of vision was the most frequent sign when the lesion was in the chiasm or optic tract.

Gross and microscopic examination showed that most of the tumors were low-grade astrocytomas and that there was an occasional oligodendroglioma or ganglioneuroma. All types had a tendency to spread toward the brain, but only rarely forward in the orbit. Exenteration of the orbital contents is rarely indicated. Surgical treatment is indicated in all cases, and a transcranial approach by frontal craniotomy is advocated. The prognosis for unilateral lesions of the optic nerve is excellent but it is less favorable in lesions of the chiasm or tracts. (9 figures, 2 tables, 15 references)

W. S. Hagler.

Gareis, Richard. **Pits in the papilla with macular cyst.** Klin. Monatsbl. f. Augenh. **132**:874-879, 1958.

Gareis reports this association of lesions in a 30-year-old man. The cyst had developed after welding without a protective glass and appeared more like a central serous retinopathy. (1 figure, 25 references)

Frederick C. Blodi.

Granström, K. O. **A family with glaucoma-like optic discs but no glaucoma.** Acta ophth. **36**:371-374, 1958.

Three sisters of late middle age exhibited extremely large excavations of the optic discs. Thorough investigation ruled out glaucoma or any other optic nerve degeneration. Two other sisters have normal eyes and three siblings are dead. It is suggested that in all three cases there is a genetically determined anomaly of the discs of no practical consequence.

John J. Stern.

Imachi, J., Kajikawa, I., Matsumoto, S. and Ito, H. **Experimental studies on chronic retrobulbar neuritis: experiments on toxic and meningeal neuritis with reference to the influences of vitamin A and B_1 deficiency on the optic nerve.** Jap. J. Ophth. **2**:143-154, May, 1958.

Vitamin A or B_1 deficiency produced Marchi balls or primary degeneration within the optic nerve. In vitamin deficient animals the administration of acetic acid caused lesions in the optic nerve and retina independent of each

other. With the administration of aspidium, there were independent lesions in the retina, optic nerve, and arachnoid, indicating that the retinal involvement does not spread to the optic nerve, the most severe lesion being in the chiasma. Vitamin A or B_1 deficiency seriously aggravates the pathohistologic changes brought about by poisoning. Circulatory disturbances (allergic arachnoiditis) markedly interferes with the function and nutrition of the optic nerve. Vitamin A or B_1 deficiency hastens disintegration of the optic myelin sheath in meningeal neuritis. Vitamin A or B_1 deficiency plays an important predisposing role in optic nerve disease. Irwin E. Gaynon.

Jaensch, P. A. **Tumors of the optic nerve.** Klin. Monatsbl. f. Augenh. **132**: 617-625, 1958.

Three girls, aged 3 to 5 years, who had unilateral glioma of the optic nerve which was excised, are described. One child apparently had neurofibromatosis. (5 figures, 34 references)

Frederick C. Blodi.

Joseph, R. and Davey, J. B. **Dominantly inherited optic atrophy.** Brit. J. Ophth. **42**:413-425, July, 1958.

The most frequent type of inherited optic atrophy is Leber's disease which has a sudden onset in adolescence and a rapid course to exceedingly poor vision. Inheritance is dominant. Pallor of the optic disc is always seen but frequently is confined to the temporal half of the disc; the amount of pallor is only an approximate guide to the degree of visual loss. Poor dark adaptation is often seen and almost all patients, both male and female present defective or absent color vision.

Two family groups are presented one of which had five affected persons in two generations and the other four. (6 figures, 4 tables, 16 references) Morris Kaplan.

Manchester, P. T. and Calhoun, F. P., Jr. **Dominant hereditary optic atrophy with bitemporal field defects.** A.M.A. Arch. Ophth. **60**:479-484, Sept., 1958.

The authors present the history of a family showing dominant hereditary optic atrophy. Some members of the family also showed bitemporal hemianopic defects. (3 diagrams, 2 tables, 9 references)

G. S. Tyner.

Petersen, Hans Peter. **Pits or crater-like holes in the optic disc.** Acta ophth. **36**:435-443, 1958.

Nine patients with crater-like holes in the optic disc are discussed. Three of them had a condition resembling central serous retinopathy, and the possibility is mentioned that this may not be a mere coincidence. Six patients had a field defect, two rather large, the others small, paracentral scotomata. No correlation was found between the size of the hole and that of the scotoma. One eye could be examined histologically and retinal edema with fibrin deposits as well as atrophy of the nerve fiber layer and the cells of the inner nuclear layer was found. (17 figures, 11 references) John J. Stern.

Quatermass, M. **Amblyopia due to ethyl alcohol.** Brit. J. Ophth. **42**:628-631, Oct., 1958.

A rare case of "toxis amblyopia" due to ethyl alcohol is described. Avitaminosis, the result of an inadequate diet, was in all probability the direct cause of the clinical picture in which the central scotoma was due to a retrobulbar neuritis. An unusual feature was the rapid response to vitamin therapy. Vision became normal within three weeks, which is considerably better than is usual in tobacco amblyopia. (2 figures and 3 references) Lawrence L. Garner.

13

NEURO-OPHTHALMOLOGY

Anastasopoulos, C., Routsonis, K. and Ierodiakonou, C. S. **Ophthalmic herpes**

zoster with contralateral hemiplegia. J. Neurol., Neurosurg. & Psychiat. **21**:210-212, Aug., 1958.

The authors describe right-sided hemiplegia with expressive aphasia which followed ophthalmic herpes zoster on the left side by four weeks. Only four similar cases could be traced in a review of the literature on focal cerebral lesions following herpes zoster. All followed herpes zoster of the ophthalmic branch of the trigeminal nerve. Their common findings and the possible pathogenesis are discussed. The view is put forward that the vessels in the neighborhood of the Gasserian ganglion may be the site of an arteritis which lead to a vascular accident in the ipsilateral hemisphere. (26 references) F. H. Haessler.

Enokson, P. and Bynke, H. **Visual field defects in arterio-venous aneurysms of the brain.** Acta ophth. **36**:568-600, 1958.

Seven cases of visual field defect occured among 38 consecutive patients with arteriovenous aneurysm of the brain. The optic radiation may be injured by direct compression or ischemia, intracerebral bleeding or hydrocephalus. Careful visual fields should be made in all cases of subarachnoid hemorrhage and migraine. (14 figures, 24 references) John J. Stern.

Heycock, J. B. and Wilson, J. **Diabetes mellitus in a child showing features of Refsum's syndrome.** Arch. Dis. Child. **33**:320-323, Aug., 1958.

Because of the great rarity of the association, the authors describe a diabetic child who exhibited most of the features of Refsum's syndrome. The latter, heredopathia atactica polyneuritiformis, is a recessive syndrome characterized by 1. atypical retinitis pigmentosa with hemeralopia and concentric constriction of visual fields, 2. a picture of chronic polyneuritis with progressive pareses of distal parts of limbs and decreased or absent reflexes, 3. ataxia and other cerebellar signs, 4. increased cerebrospinal fluid protein with normal cell count, and 5. in some cases, abnormal electrocardiographic changes, neurogenic hearing loss, pupillary abnormalities, or skin changes resembling ichthyosis. (14 references) F. H. Haessler.

Huerkamp, B. **Pupillotomy as a symptom of sympathetic irritation.** Klin. Monatsbl. f. Augenh. **133**:161-178, 1958.

Of 103 patients with Adie's syndrome, 49 percent were female; many of the patients showed other signs of increased vegetative, especially sympathetic, stimulation. (1 figure, 7 tables, 199 references) Frederick C. Blodi.

Meredith, J. M. and Adams, R. A. **Spontaneous rotary nystagmus in unilateral cholesteatoma of the cerebellopontine angle.** A.M.A. Arch. Ophth. **60**:485-487, Sept., 1958.

A case is reported in a 47-year-old white man. No other case has been found reported in the textbooks of ophthalmology. (5 references) G. S. Tyner.

Neubert, Frank R. **Complete ophthalmoplegia in acute toxic polyneuritis.** Brit. J. Ophth. **42**:632-633, Oct., 1958.

A rare case of gradually developing total ophthalmoplegia in a 19-year-old woman is described. The onset of symptoms was noted two days postpartum. Other neurologic symptoms soon followed. Cortisone therapy brought about complete recovery. (1 table, 4 references) Lawrence L. Garner.

Roughier, J., Allegre, G.-E. and Wertheimer, J. **Ocular signs of meningiomas.** J. Med. Lyon **39**:539-546, June 20, 1958.

The various types of meningiomas are described clinically, radiologically, and pathologically. Those of the suprasellar region, the olfactory groove, the lesser wing of the sphenoid, and of the orbit are discussed in detail. The ophthalmologist

has the responsibility of diagnosing these tumors in some cases and is important in the evaluation of treatment by the neurosurgeon. (6 figures)

Edward U. Murphy.

Schreck, E. **Synopsis of the visual and pupillary pathways and their damage.** Klin. Monatsbl. f. Augenh. **132**:715-720, 1958.

This summary consists of two colored plates which depict the normal anatomy of these paths and of two black and white plates which illustrate typical field defects. (4 figures) Frederick C. Blodi.

Steinvorth, E. **Retrobulbar neuritis and disturbances of other cranial nerves in the Melkersson-Rosenthal syndrome.** Klin. Monatsbl. f. Augenh. **133**:105-108, 1958.

In this syndrome recurrent facial palsies are accompanied by recurrent swellings in the face, especially of the lips and the tongue. This condition was combined with a retrobulbar neuritis on the same side in a 22-year-old man. There was also an affection of the first and third branch of the trigeminal nerve on the same side. (13 references) Frederick C. Blodi.

Thomann, Heinrich. **Rare ocular changes in syringomyelia.** Klin. Monatsbl. f. Augenh. **133**:102-105, 1958.

In a 54-year-old woman with syringomyelia a partial coloboma of one nerve head caused a scotoma. (2 figures, 6 references) Frederick C. Blodi.

14

EYEBALL, ORBIT, SINUSES

Gough, Malcolm H. **Metastatic panophthalmitis following commonplace surgical infections.** Lancet **2**:237-238, Aug. 2, 1958.

The author describes two patients in whom metastatic panophthalmitis followed pyogenic inflammation elsewhere in the body. In one of them the primary inflammation was a breast abscess and in the other paronychia. He emphasizes the importance of early and adequate antibiotic therapy. "Any patient with an acute iritis and a history of recent pyogenic infection should be suspected of having metastatic panophthalmitis." (10 references) Irwin E. Gaynon.

Kleberger, E. **Bloodless enucleation (Weihmann).** Klin. Monatsbl. f. Augenh. **132**:886-887, 1958.

This is an addition to the article by Weihmann (Arch. f. Ophth. 159:351, 1958). The author prefers the method of Myers (Am. J. Ophth. 33:1143) with the snare. (4 references)

Frederick C. Blodi.

Kleberger, E. and Stolowsky, R.-B. **An unusually large orbital cyst with marked enlargement of the bony orbit.** Klin. Monatsbl. f. Augenh. **133**:218-227, 1958.

The cyst occurred in a newborn and was repeatedly punctured. Three and one-half years later the eye on that side was markedly enlarged, highly myopic and had reduced vision (hand movements at one meter). (6 figures, 18 references)

Frederick C. Blodi.

Lemoine, Albert N., Jr. **The orbit.** A.M.A. Arch. Ophth. **60**:506-517, Sept., 1958.

This review contains 145 references.

G. S. Tyner.

Maciera, A. **Epithelization of the orbit in cases of exenteration.** Rev. brasil. de oftal. **17**:301-332, Sept., 1958.

The author reviews the classical technique of exenteration of the orbit and points out its inadequacies, particularly the slow process of epithelization of the orbit. He now uses a Thiersch graft to cover the entire orbit in all cases; recovery is greatly accelerated and the pa-

tients are more comfortable and free from postoperative complications. The author describes his technique in detail and briefly reviews the literature on the types of grafts used in exenterations. (15 figures, 11 references) Walter Mayer.

Niessen, V. **The treatment of orbital pseudotumors with local cortisone.** Klin. Monatsbl. f. Augenh. **133**:97-101, 1958.

A very chronic case of pseudotumor improved rapidly with retrobulbar cortisone injections. (3 figures, 6 references)

Frederick C. Blodi.

Walker, J. S. and Porter, G. LeR. **Transantral decompression for malignant exophthalmos.** A.M.A. Arch. Otolaryng. **68**:152-155, Aug., 1958.

The authors describe their method of orbital decompression for malignant exophthalmos which is accomplished by removal of the orbital floor through a transnasal approach. There is minimal danger of infection, the convalescence is short and there is no residual deformity. (3 figures, 5 references)

Irwin E. Gaynon.

15

EYELIDS, LACRIMAL APPARATUS

Aczel, G. **The Kettesy operation for senile entropium.** Klin. Monatsbl. f. Augenh. **132**:860-864, 1958.

The skin incision is made immediately beneath the line of lashes. The skin is mobilized and the piece of the orbicularis muscle is excised. No suture is used. This operation proved successful in ten patients. (10 figures, 2 references)

Frederick C. Blodi.

Agarwal, S. and Shrivastav, J. B. **Amyloid tumor of the eyelids.** Brit. J. Ophth. **42**:433-436, July, 1958.

Tumor-forming amyloidosis of the ocular tissues is rare and in India it is exceedingly rare. The authors describe the case of a 30-year-old woman who had noted steady, progressive swelling of both eyelids for 12 months. She had uniform swelling with a single nodule in each upper lid. The eyes themselves could not be examined and general physical examination revealed no irregularities. The nodules were excised for histologic study which revealed homogeneous amyloid tissue throughout. The etiology of this degenerative disease could not be determined but it was probably trachoma. (4 figures, 10 references)

Morris Kaplan.

Autric, C. and Carli, J. **A technique for the surgical care of entropion and trichiasis of trachoma.** Ann. d'ocul. **191**:458-462, June, 1958.

The authors make an incision over the tarsus of the upper lid and excise a long wedge of tarsus parallel to the lid margin. The tarsal edges are approximated, the lid everted and a small triangle removed near the nasal margin of the tarsus. Finally a lateral canthotomy is performed. In 216 cases there have only been two complications. (6 figures)

David Shoch.

Blodi, F. C. and Huffman, W. C. **Cicatricial ectropion caused by cutaneous blastomycosis.** A.M.A. Arch. Ophth. **59**:459-462, March, 1958.

In a 57-year-old white man with blastomycosis, the lesions responded to treatment with sulbamidine isethionate and were replaced with scar tissue. Cicatricial ectropion was treated surgically by split thickness grafts in the upper lid and full thickness grafts in the lower lid. (5 figures, 2 references) G. S. Tyner.

Friede, Reinhard. **The surgical treatment of blepharospasm.** Klin. Monatsbl. f. Augenh. **133**:270-272, 1958.

The incision is made 20 to 25 mm. from

the lateral canthus upward toward the brow. A strip of subcutis 5 mm. wide is excised. From this incision the orbicularis is incised subcutaneously along its entire circumference. Two cases were successfully treated in this way. (1 figure)

Frederick C. Blodi.

Hager, G. **Treatment of intermittent palpebral edema.** Klin. Monatsbl. f. Augenh. **133**:241-249, 1958.

Three cases of angioneurotic edema are described. In one boy it was probably due to a food allergy. A 45-year-old man responded to psychotherapy and in a 7-year-old boy a spontaneous cure occurred when he entered a hospital. (5 figures, 26 references) Frederick C. Blodi.

Krishna, N. and Lyda, W. **Acute suppurative dacryoadenitis as a sequel to mumps.** A.M.A. Arch. Ophth. **59**:350-351, March, 1958.

A case is reported which is of interest because an acute unilateral suppurative dacryoadenitis appeared in a serologically confirmed case of mumps. (1 figure, 1 reference) G. S. Tyner.

Mettier, Stacy R., Jr. **Aberrant lacrimal gland.** A.M.A. Arch. Ophth. **60**:488-491, Sept., 1958.

The author found aberrant lacrimal-gland tissue in the sclera and cornea. Similar cases in the English literature are reviewed. (4 figures, 9 references)

G. S. Tyner.

Ruben, Montague C. **Alternative to the 3-snip operation for lacrimal drainage insufficiency.** Brit. J. Ophth. **42**:626-627, Oct., 1958.

For epiphora due to poor apposition of the lower lid, as in the case of ectropion, the authors recommend the following procedure. The lower punctum is dilated and a probe inserted. A new round opening is then made about 2 mm. below the original punctum by everting the lower lid and using a 1.5 mm. trephine on the conjunctival surface immediately over the probe. The disc is cut out with scissors after being elevated with an iris forceps. After removing the probe a small piece of cotton is inserted into this hole to bring about epithelialization; it can be removed in 24 hours. If the lid seems somewhat everted the trephine opening can be made 3 mm. from the punctum. One case is cited. (2 figures, 1 reference)

Lawrence L. Garner.

16

TUMORS

Blatt, N., Ursu, A. and Popovici, V. **The invasion tendency of malignant intraocular tumors.** Klin. Monatsbl. f. Augenh. **132**:818-828, 1958.

Five malignant melanomas and two epitheliomas were examined histologically. The sclera seems to be a definite barrier against the invasive growth of a tumor. Only when the neoplasm has been in contact with the sclera for a long time will it be invaded. (13 figures)

Frederick C. Blodi.

Gaipe, M. **Contrast phase microscopy in diagnosis of ocular tumors.** Gior. ital. oftal. **10**:127-146, March-April, 1957.

After describing the appearance of sections from cases of retinoblastoma and malignant melanomas by contrast phase microscopy, the author describes in some detail the cytologic characteristics of various tumors of the eye and of its adnexa. The study was carried out on 60 patients among whom there were 38 cases of malignancy. The view is expressed that examinations of aqueous in such cases was of little value compared to examination of biopsies from the tumors themselves. (12 figures, 1 table, 93 references)

V. Tabone.

Lorenzen, U. K. **Plasmacytoma of the orbit.** Klin. Monatsbl. f. Augenh. **132**:731-735, 1958.

This tumor occurred in the right orbit of a 69-year-old woman. A biopsy was done and X-ray therapy reduced the exophthalmus. (5 figures, 23 references)

Frederick C. Blodi.

Makley, T. A., Jr. and Teed, R. W. **Unsuspected intraocular malignant melanomas.** A.M.A. Arch. Ophth. **60**:475-478, Sept., 1958.

These authors have reviewed two series of cases at the Armed Forces Institute of Pathology. One series consisted of 1,000 cases of intraocular malignant melanoma, among which were 212 eyes with opaque media (21.2 percent). The other series consisted of 969 eyes with opaque media and a history of blindness for six months or longer, 37 (3.8 percent) of which harbored unsuspected malignant melanomas. In over 11 percent of eyes with opaque media and melanoma there was no suspicion of the presence of an intraocular neoplasm. (5 tables, 6 references) G. S. Tyner.

17
INJURIES

Cuccagna, F. **Ocular manifestations following thoracic trauma.** Arch. di ottal. **62**:115-130, March-April, 1958.

On rare occasions crushing injuries of the chest cause lesions of the eye which fall into three groups: 1. optic neuritis, 2. liporrhagia of the retina, an exudative phenomenon, and 3. cyanosis retinae with fatty emboli. The author feels that these would be found more commonly if sought routinely. He states that experimental efforts to produce fatty emboli have been unsuccessful.

A case is reported in which the right eye showed evidence of thrombosis of the central artery with white lymphorrhagic patches, papilledema, and vision reduced to perception of hand movements. The left eye showed less hemorrhage, and finally improved to a visual acuity of 10/10 from the initial 3/10. The author discussed the possible relation of vena caval stasis to the cyanosis of the retina, transmitted by insufficiency of venous valves. The cause of the lymphorrhagia of the retina was considered secondary to hemorrhage or to increased intracranial pressure. One could observe turbid fat in retinal vessels, but fat was absent in the left eye. (2 figures, 49 references)

Paul W. Miles.

Levy, Walter, J. **Intra-ocular foreign body.** Brit. J. Ophth. **42**:610-616, Oct., 1958.

In a follow-up study extending from three to ten years, 272 cases of intraocular foreign body are analyzed. The anterior route of removal results in better vision than the posterior route. Six patients in ten recovered useful vision and 6.6 percent lost the eye. A point to be emphasized in anterior route removal is the necessity for maximal dilatation in order to avoid enmeshing the foreign body in the iris tissue. The eye with total cataract formation early after injury offers a poor immediate prognosis in contrast to the eye with a localized opacity. Of 34 eyes with retained intraocular foreign body, 15 required removal. Two thirds of the latter had severe infection or severe uveitis. In some instances a metallic or nonmetallic foreign body can remain inert in the eye. After use of the posterior route for removal, one third of all these eyes developed retinal detachment. The low rate of infection is ascribed to intense antibiotic therapy. (3 tables, 2 references) Lawrence L. Garner.

18
SYSTEMIC DISEASE AND PARASITES

Benedict, W. H. **Nematode and endophthalmitis.** Tennessee St. M. A. J. **51**:328-330, Aug., 1958.

In an eight-year-old girl the painless blind eye with leukocoria was enucleated because the clinical picture suggested a diagnosis of retinoblastoma. Studies of the enucleated eye revealed a granuloma in which eosinophiles were conspicuous, which was the result of infection with a dog ascarid, Toxocara canis.

Irwin E. Gaynon.

Björk-von Bahr, S. **Is the determination of the retinal blood pressure of value in modern treatment of hypertension?** Acta ophth. **36**:536-539, 1958.

There seems to be no special advantage in determining the retinal blood pressure during treatment of hypertension by ganglion-blocking agents; the cerebral circulation varies with the general circulation. (2 figures, 5 references)

John J. Stern.

Cohen, E. I. **Thyrotropic exophthalmos: clinical forms and neurologic aspect.** Presse Med. **66**:1040-1043, June 11, 1958.

The author distinguishes two clinical forms of exophthalmos associated with thyroid disease: the active and the residual. Hypophyseal inhibitors, X-ray therapy, or orbital decompression may be indicated in the former but definitely not in the latter. The diagnosis of the inactive form is suggested when the patient's chief complaint concerns the appearance of the protruding eyes. (2 figures) Edward U. Murphy.

Cramer, F. K. and Lamela, N. A. **The fluorescein permeability test in arterial hypertension.** Arch. oftal. Buenos Aires **33**:65-69, March, 1958.

The permeability of the blood-aqueous barrier was examined with Amsler and Huber's test in 38 patients with high blood pressure. An abnormally rapid appearance and a moderate to marked increase in the concentration of the dye was recorded in most cases. However, no demonstrable relationship in degree existed between such findings and the severity of the hypertension, the severity of the ophthalmoscopic picture or the fact that renal function were more or less handicapped. (5 graphs, 8 references)

A. Urrets-Zavalia, Jr.

Duncan, L. J. P., MacFarlane, A. and Robson, J. S. **Diabetic retinopathy and nephropathy in pancreatic diabetes.** Lancet **1**:822-826, April 19, 1958.

Diabetic retinopathy has been considered, like Kimmelstiel-Wilson bodies in the kidneys, a pathognomonic complication of diabetes, related to irregular elevations of blood sugar. However, diabetes is to some degree a gene-determined disease. As such, the appearance of microaneurysms in body capillaries could be gene-determined, and not due to an elevation of blood sugar. There are two possible types of diabetes, one gene-determined and associated possibly with an anti-insulin mechanism, and another secondary to inflammatory or degenerative disease of the islets in the pancreas. Few cases of the latter type have been published.

In this paper, the case of diabetes in an architect who died at the age of 48 years is thoroughly described. There was no family history of diabetes. The patient had a severe generalized mumps in 1920, followed by diabetes mellitis in 1928 which required from 28 to 40 units of insulin daily. In 1936 he developed steatorrhea, loss of weight, loss of ankle and knee reflexes, early diabetic retinopathy, and nephritis. The kidney "infections" became so bad that in 1957 the ureters were transplanted into the ileum. He died a few months later with uremia. At death there was moderately severe diabetic retinopathy (drawings showing microaneurysms, round hemorrhages, and discrete exudates, no pathologic sections). The heart was normal. There was gross

pancreatic fibrosis with reduced islet tissue.

The conclusion was that capillary microaneurysms in various parts of the body are due to the elevation of blood sugar and are not gene-determined. It seems reasonable to suppose that these complications would not occur if the carbohydrate metabolism could be perfectly regulated. (7 figures, 16 references)

Paul W. Miles.

Ferguson, R. H., and Paris, J. **Sarcoidosis: a study of twenty-nine cases, with a review of splenic, hepatic, mucous membrane, retinal, and joint manifestations.** A.M.A. Arch. Int. Med. **101**:1065-1084, June, 1958.

The incidence of sarcoidosis in Army Induction Centers has been about two in 100,000. In an Army hospital it was 0.12 percent of total admissions. In this paper, 29 cases are reported, of which 26 occurred in negroes and 24 in subjects from the southern United States. Many patients were asymptomatic, ten had cough, six had blurred vision, six lost weight, five had fever, five dyspnea, four enlarged lymph nodes, and four had chest pain. Twenty-four showed enlarged mediastinal lymph glands, 18 peripheral lymphadenopathy, and 19 showed evidence of lesions of the lung parenchyma. The test found most satisfactory for diagnosis was biopsy of the scalene fat pad gland.

There was anterior uveitis in five subjects, one of whom showed a retinitis without choroiditis; the skin was involved in three, the joints in three, and the nasal mucosa was atrophic in one. Tuberculin and histoplasmin tests were usually negative, and the blood picture was usually normal. The sedimentation rate proved to be a fairly good indication of the activity of the disease.

Anterior uveitis in these cases consisted of iritis with gray keratic precipitates on the corneal endothelium. These were said to clear within a few weeks on topical cortisone drops. The one case with retinitis is illustrated by photographs. There were yellowish white nodules on the retinal vessels and perivenous streaks. Evidently there are only eight cases of sarcoid retinitis reported in the literature. Previous reports have described periphlebitis and occlusion of vessels with epithelioid granulomas.

No internal treatment was advised, except in patients with splenomegaly or hematopenia. (2 figures, 5 tables, 62 references) Paul W. Miles.

Juurikkala, Anita. **Toxoplasmosis.** Acta ophth. **36**:580-584, 1958.

In 37 cases observed, the disease was congenital in 16 patients, acquired in four, due to rupture of a pseudocyst in 15 and of obscure onset in two. The most common symptom (in 35 cases) was a chorioretinal focus or scar. Strabismus was present in 18 cases, microphthalmus in seven, and nystagmus in five. Uveitis was seen in three patients and iritis in two. The dye test titer was between 1 : 64 and 1:16384. Symptoms in organs other than the eyes were insignificant. Diagnosis is determined by the clinical picture and serologic reactions; either alone is not adequate. Diaminopyrimidine (Daraprim) is the most effective drug for the treatment of toxoplasmosis; sulfadiazine has also given good results, and a combination of both is recommended because of the synergistic effect they have. (2 to 4 gm. of sulfadiazine, 25 to 50 mg. Daraprim daily); both dosages are cut down to half one to three weeks later. (3 tables, 14 references) John J. Stern.

Muzzio D'Amelia, J. C., Balina, L. M., Nicoli, C. and Nallar, L. **Ectodermosis erosiva pluriorificialis due to phenylbutazone administration: report of a case treated with ACTH.** Arch. oftal. Buenos Aires **33**:79-86, March, 1958.

In a 52-year-old man who has had muscular and articular aches of a seemingly rheumatoid nature, ectodermosis erosiva pluriorificialis (erythema exudativum multiforme, Hebra; Stevens-Johnson's syndrome) appeared a few hours after the ingestion of two 0.20 g. tablets of phenyl-butazone. The disease followed a chronic course, with periodic ameliorations and relapses of variable severity. At the end of six months, scattered atrophic areas were found in the skin and most mucous membranes, together with widespread infarction of the lymph nodes. The conjunctiva was dry and congested, and diffuse, superficially vascularized corneal opacities were present; the lacrimal secretion was very scarce and mucous filaments were seen to adhere to the corneal surface. Administration of daily doses of 20 U. of ACTH for six weeks resulted only in subjective improvement. (2 figures, 52 references)

A. Urrets-Zavalia, Jr.

Pietruschka, G. **Hypotony of the globe in systemic disorders.** Klin. Monatsbl. f. Augenh. **132**:839-847, 1958.

Routine tonometry was done on 1,590 patients on medical wards; 178 had a pressure less than 12 and out of these only 35 patients had a pressure of less than 10 mm. Hg. Hypotony is usually due to a desiccation of the eye leading to a hyposecretion of aqueous. It occurs more frequently in diseases of the heart, anemia, and vascular insufficiencies. (7 tables, 36 references) Frederick C. Blodi.

Rebello, N., de Castro, J. and Alcover, P. **Ocular leishmaniasis, report of a case.** Rev. brasil. de oftal. **17**:279-298, Sept., 1958.

The authors describe in great detail the clinical findings in a patient with leishmaniasis. The right eye was very seriously affected, while the left eye had only minor eye symptoms. Because of very marked symblepharon and extreme vascularization this patient required surgery in three different stages before a fairly satisfactory cosmetic result could be obtained. During the first operation only lysis of the symblepharon and of all the adhesions in the cul de sac was attempted. During the second operation a repeated lysis of adhesions was combined with an enucleation. During the third and final stage the orbital cavity was cleaned of all granulation tissue. Each of these operations was extremely difficult because of the large amount of hemorrhage encountered.

The authors briefly discuss the world literature on ocular leishmaniasis as well as its geographic distribution. (14 figures, 18 references) Walter Mayer.

Reed, H., Lindsay, A., Silversides, J. L., Speakman, J. and Rees, D. L. **The uveoencephalitic syndrome or Vogt-Koyanagi-Harada disease.** Canad. M. A. J. **79**:451-459, Sept. 15, 1958.

The authors review the pertinent literature and describe the clinical manifestations of this syndrome. They present five rather extensive reports of cases and discuss them in relation to the data in their introductory information. They discuss the importance of the viral theory, sympathetic ophthalmia, and allergy in a consideration of the etiology. (2 figures, 25 references) F. H. Haessler.

Remler, O. **Two cases of external ophthalmomyiasis.** Klin. Monatsbl. f. Augenh. **133**:83-86, 1958.

In both instances the larvae were in the conjunctival sac. The larvae are usually multiple and hidden in folds. (4 references) Frederick C. Blodi.

Stadler, H. E. **Familial dysautonomia.** J. Pediat. **53**:481-483, Oct., 1958.

The only ocular disturbance in an eight-year-old boy with familial dysautonomia

was an absence of tears. He was dry-eyed when crying. (2 figures, 3 references)

Irwin E. Gayon.

Thomas, P. K. and Smith, E. B. **Ocular manifestations in idiopathic hyperlipemia and xanthomatosis.** Brit. J. Ophth. **42**: 501-506, Aug., 1958.

A patient is described who had ocular findings other than the palpebral xanthomata and arcus lipoides cornea which are commonly associated with idiopathic hyperlipemia. The fundus revealed scattered yellow areas of all sizes throughout; there was one area of retinal detachment. Small yellowish nodules were also noted on the iris. Since the central area was involved, vision was reduced. Heparin was of no value in this case. (3 figures, 9 references)

Lawrence L. Garner.

Watzke, Robert C. **Marfan's syndrome.** A.M.A. Arch. Ophth. **60**:492-497, Sept., 1958.

A kinship of 41 persons in four generations is outlined; 24 of them have ocular manifestations of Marfan's syndrome. Aortic aneurysms and other aortic anomalies have been shown to occur frequently in these patients. (1 diagram, 1 table, 22 references)

G. S. Tyner.

Worthen, H. G. and Good, R. A. **The de Toni-Fanconi syndrome with cystinosis: clinical and metabolic study of two cases in a family and a critical review on the nature of the syndrome.** A.M.A. J. Dis. Child. **95**:653-688, June, 1958.

In this long article the authors add to evidence that cystinosis is regularly a part of the de Toni-Fanconi syndrome. Part of the diagnosis consists of slitlamp observation of cystine crystals in the cornea. The syndrome was defined as a vitamine D resistant hypophosphatemic rickets, with acidosis, glycosuria, hyper-amino aciduria, and organic aciduria due to deficient reabsorption of phosphorus, amino acids and glucose in the proximal convoluted tubules of the kidneys. Other amino acids accumulate in the body, but do not crystallize. The cystine also accumulates in other body tissues, particularly the reticulo-endothelial system. The etiology is unknown, but the biochemical blood and urine relations are well established. Of the two brothers studied here, one died and was autopsied. The other has stunted growth, but the disease seems to be under control.

Paul W. Miles.

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CONGENITAL DEFORMITIES, HEREDITY

Cambiasso, Raúl Héctor. **Intrafamilial occurrence of keratoconus and myopia.** Arch. oftal. Buenos Aires **33**:123-128, May, 1958.

One case of manifest keratoconus, three of myopia ranging from −0.50 to −14 D, and two of "keratocône frustre" associated with myopia of a variable degree, were observed in a family of nine in three generations. The conclusion is drawn that a close essential relationship must exist between both conditions, which would be the result of the polyphenic action of the affected gene; yet, the possibility that two pathologic genes could have come into play should be borne in mind. (1 figure, 1 table, 2 references)

A. Urrets-Zavalia, Jr.

Gaertner, J. **The pathogenesis and therapy of the glaucoma in Marchesani's syndrome.** Klin. Monatsbl. f. Augenh. **133**:31-93, 1958.

Two sisters, 14 and 17 years of age, with Marchesani's syndrome (spherophakia and brachydactyly) also had glaucoma, a frequent complication. The chamber angle is narrow or closed in these eyes. Miosis will lead to a pupillary block because of the round lens. Mydriasis or lens extraction is the treatment of choice. (4 figures, 6 references)

Frederick C. Blodi.

Hollwich, F. **Passow's operation in chemical and thermal burns of the eye.** Med. Klin. **53**:1459-1462, Aug. 22, 1958.

Incision of the conjunctiva at the limbus and undermining it is indicated whenever there is a subconjunctival transudate or exudate as a result of a chemical or thermal burn. If the cornea is similarly injured its epithelium should be scraped off. (2 tables, 19 references)

F. H. Haessler.

Keerl, G. **Congenital cornea-lens synechia with glaucoma.** Klin. Monatsbl. f. Augenh. **133**:50-59, 1958.

Two patients who have this congenital anomaly are described. Glaucoma is a nearly constant complication. (6 figures, 30 references) Frederick C. Blodi.

Koskenoja, M. **Eye findings in children born of diabetic mothers.** Acta. ophth. **36**: 559-564, 1958.

Five out of 49 children of diabetic mothers, between two and six years old, showed pathologic findings; two had bilateral cataract, and one of these had nystagmus as well, and the other had strabismus; two others had hypertelorism, associated in one of them with strabismus and in the other with optic atrophy; the fifth child had a unilateral uveal coloboma. Three of these five patients were mentally retarded. (3 figures, 23 references) John J. Stern.

Tosch, Cäcilie. **A pedigree of floriform cataract.** Klin. Monatsbl. f. Augenh. **133**:60-66, 1958.

Members of five generations of a family were examined. The cataracts were found in three generations. There were ten affected and ten normal members (dominant inheritance). In the fourth generation the children were less than seven years old and this type of cataract develops somewhat later. (4 figures, 29 references) Frederick C. Blodi.

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HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Dansey-Browning, G. C. **Ophthalmic disease in Hong Kong.** Brit. J. Ophth. **42**:394-401, July, 1958.

Hong Kong consists of 193 islands with a total of 62 square miles of inhabitable or otherwise useful area. The population is greater than two and a half million, 98 per cent being Chinese with two million living in the 12 square miles of urban Hong Kong. In the slum areas disease is rampant and therapy of the ancient herb medicine of the East is very frequently sought. In 1953 the incidence of ophthalmic disease among the poor was 150,000 cases per year. The principal diseases seen at the clinics are acute affections of the anterior segment due mostly to malnutrition, phlyctenular disease, syphilis, trachoma, cataract, and glaucoma. The conditions leading to such extensive numbers of diseased persons are the tremendous overcrowding, poor sanitation, inadequate diet, and the large incidence of syphilis. There are about 3,000 blind in Hong Kong of which half became blind before the age of ten years, presumably because of malnutrition. Trauma, except for damage by fireworks, is minimal because there is little heavy industry in the colony.

The government eye clinics are expanding rapidly and improved ophthalmic conditions are expected. Most of the surgery for cataract is done on outpatients who are sent home immediately after. The incidence of complications seems to be no greater than in those fully hospitalized. (3 tables, 4 references)

Morris Kaplan.

Geppert, Maria-Pia. **The importance of statistical methods for biological processes and medical knowledge.** Klin. Monatsbl. f. Augenh. **133**:1-14, 1958.

After a brief discussion of the concepts of "chance" and "objective, mathematical probability" the meaning of a chance-critical method in biologic and medical research is explained. This method is indispensable if results obtained from a limited number of observations are to be extended to larger, unknown populations. (5 figures) Frederick C. Blodi.

Mercier. **A contribution towards the study of ocular fatigue.** Bull. et mém. Soc. franç. d'opht. **70**:540-555, May, 1957.

The contributing factors in ocular fatigue, one of the most annoying problems of modern living, are analyzed. Only persons with normal visual acuity and apparently emmetropic refraction were investigated. The components and constituents of possible eye-strain under various visual tasks and in more or less difficult situations were studied in detail. The patients were divided into four groups. Group I consisted of persons who worked under fluorescent lights for many hours; Group II of those who watch or produce television performances; Group III of radar operators; and group IV of pilots and aeronautic personnel. After discussing the various influential elements and separating them as to their causes and effects suggestions were made how to combat and relieve specific individual complaints. (33 references)

Alice R. Deutsch.

Nomura, Atsuko. **Experimental studies on twilight vision and road safety.** Jap. J. Ophth. **2**:134-143, May, 1958.

When exposure to intense illumination is less than one second, the change in visual performance is small. When the environmental illumination intensity is bright (auto lights on), the glare is lessened upon exposure to light stimulation.

Visual perception after prolonged exposure to light is better in the case of binocular vision than it is in the monocular.

Tinted windshields decrease visual perception at night, but decrease the amount of glare and head absorption to a greater degree. Visual perception was satisfactory for blue, green and red lights, while that for yellow was poor. (3 figures, 1 table, 7 references) Irwin E. Gaynon.

Vola, Jean. **Survey on trachoma in elementary school in Saint-Louis du Sénégal (French West Africa).** Rev. intern. du trachome **35**:29-44, 1958.

The author, after giving an account of the means of investigation employed (systematic examination with a biomicroscope, importance of laboratory work), correlates the statistical results of an inquiry into a group of elementary schools at Saint-Louis du Sénégal. This inquiry was carried out among 3,143 pupils, grouped according to age in various schools.

The average index of trachoma is 18.32 percent. This estimate goes up to 20.1 percent after elimination of the European pupils, only African children being then considered.

A short epidemiologic study of environment (geography, climate, geology, dwellings), gives an explanation of the important variations of the index which ranges from 10 percent to 41.5 percent in the poorest districts.

With regard to morphology, the author points out the frequency of stage I. It is a silent stage which can be observed with the biomicroscope and leaves the eye apparently sound.

The agents of dissemination (sand, flies, overcrowded dwellings) are those already known in all trachoma-affected countries and it does not seem as if modern Africa were to escape the problems set by this pre-eminently social disease. (3 tables) Author's summary.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

DEATHS

Dr. Henry Bassett Lemere, Laguna Beach, California, died October 6, 1958, aged 86 years.

Dr. Morton Thelman Siegel, West Coniva, California, died September 15, 1958, aged 36 years.

Dr. Francis Stenard Schwarz, Jr., Cleveland, Ohio, died August 31, 1958, aged 37 years

ANNOUNCEMENTS

NEW YORK EYE AND EAR MEETING

The annual spring meeting of the alumni association of the New York Eye and Ear Infirmary will take place April 20th to 23rd, at the infirmary.

Symposia will be offered on headache, complications of ocular surgery at the operating table and their management, management of postoperative complications (24 hours to two weeks), practical application of new therapeutic measures in ophthalmology, and ophthalmic plastic surgery. There will also be a closed-circuit television demonstration of surgical operations.

In addition, courses will be offered on the following subjects:

Recent advances in biomicroscopy, contact lens fitting, external eye diseases, indirect ophthalmoscopy, pterygium surgery, tonography, vertical eye muscles, recent advances in ophthalmic research, ocular pathology, macula function, orthoptics, electrophysiology of the eye, and corneal transplants.

Additional information may be obtained by writing to Dr. John R. Finlay, secretary, Alumni Association, 218 Second Avenue, New York 3, New York.

PLASTIC SURGERY COURSE

A three-week intensive course in ophthalmic plastic surgery will be conducted in New York April 23rd to May 15th.

The course will consist of lectures, sessions in doctors' offices, preliminary and follow-up cases that are operated on during the time of the course, moving picture demonstrations of various ophthalmic plastic procedures, observation and assistance at the operating table on actual surgical cases, cadaver work and lectures and demonstrations, ancillary subjects such as photography, pathology, and X ray and radiation.

The course will be limited to eight students and well be given at the New York Eye and Ear Infirmary, Manhattan Eye, Ear, and Throat Hospital and the Hempstead General Hospital. The fee will be $250.00.

Anyone interested should contact the Registrar of the New York Eye and Ear Infirmary Post-Graduate School or any of the following:

Dr. Wendell L. Hughes, Hempstead, New York; Dr. Byron C. Smith, New York City; Dr. J. Gordon Cole, New York City.

MISCELLANEOUS

CLEVELAND CLINIC COURSE

The Frank E. Bunts Educational Institute affiliated with the Cleveland Clinic Foundation gave a postgraduate course in ophthalmology during December. Guest speakers were Dr. J. Gordon Cole, New York, Dr. P. Robb McDonald, Philadelphia, and Dr. Benjamin J. Wolpaw, Cleveland. Participating faculty from the Frank E. Bunts Educational Institute were Dr. George H. Curtis, Dr. Roscoe J. Kennedy, Dr. Charles L. Leedham, Dr. George Lockhart, III, Dr. Lawrence J. McCormack, Dr. James E. Nousek, Dr. William L. Proudfit, Dr. Carl E. Wasmuth, and Dr. Guy H. Williams, Jr.

FIGHT FOR SIGHT AWARDS

The Scientific Advisory Committee of the National Council to Combat Blindness, Inc., has announced its 1958-1959 Fight for Sight awards totaling $128,214 in grants-in-aid and fellowships allocated to medical colleges, hospitals, and individual investigators, for research in ophthalmology and related sciences.

Many of the grants represent initial support by the organization to basic research investigations in ophthalmology; others will provide continued support to projects which have received council aid in the past.

This action by the Scientific Advisory Committee provides 34 grants-in-aid to medical colleges and hospitals in the United States and abroad, and a special award to the New York Academy of Sciences for an International Symposium on Photoreception, in addition to eight full-time research fellowships, one traveling fellowship, and 21 student research fellowships.

Five foreign medical centers share in the grant-in-aid awards. They are: General Hospital, Mexico City, Mexico; Hadassah University, Department of Ophthalmology, Jerusalem, Israel; Kumamoto University Medical School, Japan; Medical Research Council, Institute of Ophthalmology, University of London, England; and, Nuffield Laboratory of Ophthalmology, Oxford University, Oxford, England.

Fight for Sight grants-in-aid and full-time research fellowships are generally awarded for a period of one year and may be extended for an additional period with the approval of the Scientific Advisory Committee of the National Council to Combat Blindness—chairman, Charles A. Perera, M.D., associate clinical professor of ophthalmology, College of Physicians and Surgeons, Columbia University.

The council announces that it is now accepting applications for its 1959-1960 Fight for Sight grants-in-aid, research fellowships, and summer (1959) student fellowships.

The closing date for receipt of completed applications for grants and fellowships has been advanced to February 1, 1959, in order to allow ample time for processing and review. Applications for summer student fellowships will be reviewed in advance of the June meeting of the Scientific Advisory Committee. Appropriate forms may be obtained by addressing: Secretary, National Council to Combat Blindness, Inc., 41 West 57th Street, New York 19, New York.

The National Council to Combat Blindness' Scientific Advisory Committee which votes on all awards, now numbers 18, having recently welcomed to its ranks a new member, Frank W. Newell, M.D., chairman of the Section of Ophthalmology, Department of Surgery, University of Chicago Medical School. Other members of the committee are:

James H. Allen, M.D., chief, Department of Ophthalmology, Tulane University, School of Medicine; Bernard Becker, M.D., chief, Department of Ophthalmology, Washington University School of Medicine; Alson E. Braley, M.D., director, Department of Ophthalmology, State University of Iowa, University Hospitals; Frederick Crescitelli, Ph.D., Department of Zoology, University of California; Arthur G. DeVoe, M.D., chief, Department of Ophthalmology, New York University-Bellevue Medical Center; Dan M. Gordon, M.D., assistant professor, Department of Ophthalmology, New York Hospital-Cornell Medical Center; Charles Haig, Ph.D., associate professor Department of Physiology and Pharmacology, New York Medical College; Michael J. Hogan, M.D., director, Francis I. Proctor Foundation, University of California Medical School; Peter C. Kronfeld, M.D., professor, Department of Ophthalmology, University of Illinois Medical School; Irving H. Leopold, M.D., director of research, Wills Eye Hospital, Philadelphia; A. E. Maumenee, M.D., director, Department of Ophthalmology, The Johns Hopkins University Medical School; Stuart Mudd, M.D., professor, Department of Microbiology, University of Pennsylvania School of Medicine; Theodore C. Ruch, Ph.D., chairman, Department of Physiology and Biophysics, University of Washington; Samuel L. Saltzman, M.D., assistant professor, Department of Ophthalmology, New York Medical College; Kenneth C. Swan, M.D., chief, Department of Ophthalmology, University of Oregon; Phillips Thygeson, M.D., professor of Ophthalmology, University of California Medical School.

FIGHT FOR SIGHT GRANTS-IN-AID

Adler, Francis H., and Frayer, William C. (continuation), University of Pennsylvania, Philadelphia, "Study of the factors involved in the proliferation of the retinal pigment epithelium in disease," $1,000.

Alpern, Mathew, University of Michigan School of Medicine, Ann Arbor, "The psychophysiology of the photopupillary mechanism," $3,600.

Auerbach, Edgar (continuation), Hadassah University Hospital and Medical School, Jerusalem, Israel, "Light and color perception: Participation and specification of receptor mechanisms in normal and pathologic conditions," $4,500.

Balazs, Endre A. (continuation), Massachusetts Eye and Ear Infirmary, Boston, "Studies on the hyaluronic acid formation of the vitreous body," $4,000.

Breinin, Goodwin M. (continuation), New York University Post-Graduate Medical School, Bellevue Medical Center, New York, "Electromyography of the extraocular muscles including stimulation studies and evaluation of drug effects on the action current," $2,500.

Burns, Robert P. (continuation), College of Physicians and Surgeons, Columbia University, New York, "Tissue culture of cytomegalic inclusion disease virus with electron microscope studies," $1,850.

Cibis, Paul A. (continuation), Washington University School of Medicine, St. Louis, "Histopathology of the eye with oblique illumination," $4,000.

Donn, Anthony, College of Physicians and Surgeons, Columbia University, New York, "The active transfer of sodium across the corneal epithelium," $1,800.

Eliasson, Sven G. (continuation), University of Texas, Southwestern Medical School, Dallas, "The role of visual impulses in the control of eye muscle activity," $5,000.

Ellerbrock, V. J. (continuation), Ohio State University, Columbus, "Preparation of textbook on partial sightedness," $250.

Fisher, Earl, Jr., Tulane University, School of Medicine, New Orleans, "Mechanism of corneal damage following certain bacterial infections," $2,160.

Garron, Levon K., and Foerster, Helenor Campbell (continuation), University of California Medical School, San Francisco, "Special histopathologic study of endogenous uveitis," $4,000.

Goldberg, Bernard, and Tabowitz, David, Dept. of Research, Division of Radioisotopes, New York Eye and Ear Infirmary, New York, "Development of improved instrumentation using radioisotope techniques in the clinical differentiation of malignant melanomas from inflammatory lesions of the anterior ocular segment," $3,000.

Green, Harry (continuation), Wills Eye Hospital, Philadelphia, "Elaboration of the bicarbonate

ion in aqueous humor and its relation to the dynamics of aqueous flow." $5,500.

Hanna, Calvin, College of Medicine University of Vermont, Burlington, "Studies on cataract formation: Nucleic acid changes in the normal and radiated eye," $3,000.

Hepner, Ray (continuation), University of Missouri School of Medicine, Columbia, "The effects of material hyperoxemia on the intrauterine development of the eye," $1,000.

Jacobson, Jerry Hart (continuation), New York Eye and Ear Infirmary, New York, "Differential electroretinography in clinical ophthalmology," $1,500.

Jampolsky, Arthur, Stanford University Hospital and School of Medicine, San Francisco, "Study of eye movements by high speed photography," $2,000.

Kirber, Maria W., Women's Medical College of Pennsylvania, Philadelphia, "Experimental viral uveitis," $2,500.

Langham, Maurice E., Institute of Ophthalmology, University of London, "Regulation of the intraocular pressure," $1,200.

Maumenee, A. Edward, Johns Hopkins University School of Medicine, Baltimore, "Antigen-antibody reactions in the rabbit cornea," $5,000.

Michaelson, I. C. (continuation), Hadassah University Medical School, Jerusalem, "(A) Mucopolysaccharide content of vitreous humor in normal and abnormal conditions. (B) Mucopolysaccharide content of the conjunctival secretion in certain diseases of the conjunctiva," $2,000.

Mitsui, Yukihiko, Kumamoto University Medical School, Honjomachi, Japan, "Prevention of epidemic keratoconjunctivitis by vaccination," $1,200.

Pirie, Antoinette, Oxford University, Oxford, England, "Study of changes in the constituents, metabolism and histology of the lens during development of cataract," $3,000.

Sloan, Louise L., Johns Hopkins University Medical School, Baltimore, "Clinical significance of the AC/A ratio," $2,646.

Smelser, George K. (continuation), College of Physicians and Surgeons, Columbia University, New York, "Relations of the exophthalmogenic and thyrotropic pituitary principles," $900.

Solanes, M. Puig (continuation), General Hospital, Mexico City, Mexico, "Comparative anatomic and physiologic study of the structures related to the maintenance of intraocular pressure in Mexican Indians and white people," $961.

Thornfeldt, Paul R., Good Samaritan Hospital Research Foundation, Portland, Oregon, "An investigation of the presence of hyaluronidase sensitive mucopolysaccharide in tissues of filtration angle of fetal and infantile eyes," $675.

Wilson, Fred M., Indiana University, Medical School, Indianapolis, "Evaluation and localization of possible glaucomatogenic effects of experimental beta irradiations of the limbus," $1,500.

Wolken, Jerome J. (continuation), University of Pittsburgh Medical School, Pittsburgh, "Photoreceptor structures," $2,500.

Zimmerman, Lorenz E., Armed Forces Institute of Pathology, Washington, D.C., "Pathology, pathogenesis, prevention, and treatment of fungus infections of the eye," $5,150.

Fight for Sight special grants

11-cis isomer of vitamin A in the prevention of retinal rod degeneration, $500. To aid in the preparation and presentation of a paper on this study which was initiated in 1955 under a Fight for Sight Research Fellowship to Albert B. Chatzinoff, M.D., at Mount Sinai Hospital, New York.

Trachoma mobile unit, $7,500. To aid the Department of Ophthalmology of the Hadassah University Medical School, Jerusalem, Israel, in extending its trachoma control program through examination and treatment to the populations in outlying sections where such medical care is inaccessible. This unit, under the direction of I. C. Michaelson, M.D., is being created with the view that it will serve as a model for the establishment of other such units in that area of the world.

International Symposium on Photoreception, $850. To underwrite a portion of the cost of organizing and conducting a Symposium on Photoreception held on January 31 and February 1, 1958, under the auspices of the New York Academy of Sciences, Biology Section, Jerome J. Wolken, Ph.D., of the Biophysical Research Laboratory, Eye and Ear Hospital, University of Pittsburgh, who has been engaged in the study of photoreceptor structures of the eye with the aid of Fight for Sight Awards, served as general chairman of the symposium.

Studies in amblyopia and glaucoma, $672. To Walter Kornblueth, M.D., Hadassah University Medical School, for travel costs to enable the investigator to spend one year at the Stanford University School of Medicine, Department of Ophthalmology, in full time research with Arthur Jampolsky, M.D., Milton Flocks, M.D., and Earl McBain, M.D.

Fight for Sight fulltime research fellowships

Barishak, Robert Y., University of California, School of Medicine, San Francisco (continuation), "Tissue culture of malignant melanomas," $3,600.

Brandt, Philip W., College of Physicians and Surgeons, Columbia University, New York, "Study of the cytologic mechanism of fluid transport through the membranes in the anterior segment of the eye by methods of light and electron microscopy," $3,400.

Holmberg, Ake Siguard, Washington University, School of Medicine, St. Louis, "Ultrastructure of the ciliary epithelium and its connection with secretion of aqueous humor," $5,000.

Krishna, Narendra, University of Pennsylvania, Graduate School of Medicine, Philadelphia (continuation), "Electromyography and related electrophysiologic techniques in ophthalmology," $4,000.

Tokunaga, Tsugihiko, University Hospitals, State University of Iowa, Iowa City, "I. Cytologic

study by electron microscopy on radiation cataract." "II. Study on incipient opacities of experimental cataracts," $4,800.

Wania, Jamshed H., Wilmer Institute, Johns Hopkins University Medical School, Baltimore, "Studies in neuro-ophthalmology," $700.

Waton, Nicholas G., Tulane University School of Medicine, New Orleans, "The nature of central and peripheral synaptic transmission in the optic pathway," $5,000.

FIGHT FOR SIGHT SUMMER STUDENT FELLOWSHIPS

Barasch, Kenneth Robert, New York Hospital-Cornell Medical Center, New York, "Effects of blood plasma volume and osmolality on intraocular pressure," $600.

Crawford, William F., University of California School of Medicine, San Francisco, "Research on mast cells including differential stains and sequential gross studies." $600.

Ernest, J. Terry, University of Chicago, Chicago, "Dynamics of water transport in the eye," $600.

Giles, Kenneth M., University of Oregon Medical School, Portland, "Transport of glucose across the lens surface," $600.

Henkind, Paul, New York University-Bellevue Medical Center, New York, "Study of Descemet's folds in diabetic and normal human subjects," $600.

Innes, James Walker, New York Hospital-Cornell Medical Center, New York, "Study on the physiology of corneal elasticity as related to certain ocular disease states," $600.

Katz, Joseph, The University of the State of New York, Brooklyn, "Use of acrylics of the nonpolymerizing type in restoration of the enucleated and eviscerated socket," $600.

Madison, James B., Tulane University School of Medicine, New Orleans, "Determination of the effects of hemicholinium upon intraocular pressure," $600.

Muskat, David, University of Pittsburgh Medical Center, Pittsburgh, "Problems in photoreception related to the problems of vision, energy transfer, and photosynthesis," $400.

O'Brien, James Edward, University of Vermont College of Medicine, Burlington, "Intracellular biochemical changes in the gamma-ray treated eye," $600.

Rubin, Laurence Edward, Washington University School of Medicine, St. Louis, "Diabetic retinopathy," $600.

Sabey, Andrew Charles, The University of the State of New York, Syracuse, "1. Experimental and clinical study of postoperative wound hemorrhage in ophthalmic surgery"; "2. Effects of iproniazed (Marsilad) and related amine oxidase inhibitors on intraocular pressure," $600.

Shepherd, Gordon Murray, Harvard Medical School, Boston, "A study of the influence of visual cortex on the cortical control of movement," $600.

Srebro, Richard, Washington University School of Medicine, St. Louis, "A stress-strain curve for sclera," $600.

Starr, Wilson Clayton, Duke University School of Medicine, Durham, North Carolina, "Evaluation of results of surgery in glaucoma," $600.

Stiles, James Fleming, College of Medicine, State University of Iowa, Iowa City, "Distribution in the rabbit's eye of C^{14}-labelled compounds," $600.

Stobbe, Juergen A., University of Louisville School of Medicine, Louisville, Kentucky, "Action of lyophilized vitreous on experimentally produced hematogenous opacities in the rabbit vitreous," $600.

Traugh, George Holton, Jr., Medical College of Virginia, Richmond, "Clinical and experimental study on the incidence of eye disorders in sickle-cell anemia and hemoglobin-C disease," $600.

Tsang, Wallace, University of California School of Medicine, San Francisco, "Study development of angle of anterior chamber in embryo and fetal eyes with use of carbohydrate and trichrome stains," $600.

Varriale, Philip, The University of the State of New York, Brooklyn, "Pleoptics in management of strabismus," $600.

Young, Robert Rice, Marine Biological Laboratory, Woods Hole, Massachusetts, "A study of the energetics of generator potentials," $500.

SOCIETIES

NASSAU MEETING

At a recent meeting of the Nassau (county, New York) Ophthalmological Society, Dr. Girolamo Bonaccolto, New York, presented a paper on "Dacryocystorhinostomy." The next meeting of the society will be on February 24th.

BROOKLYN MEETING

The 150th regular meeting of the Brooklyn Ophthalmological society was held on November 24, 1958. During the scientific program, "The management of complicated therapeutic problems," was discussed, with Dr. Frederick H. Theodore acting as moderator, and the following guest speakers participating: Dr. Irving H. Leopold, Dr. Abraham Schlossman, Dr. George N. Wise, and Dr. Samuel Weinstock.

CENTRAL ILLINOIS OFFICERS

Newly elected officers to serve during 1959 for the Central Illinois Society of Ophthalmology and Otolaryngology are: President, William F. Hubble, Decatur; president-elect, Charles D. Sneller, Peoria; vice-president, Edgar T. Blair, Springfield; delegate at large, Leroy Porter, Urbana; secretary-treasurer, Clarence A. Fleischli, Springfield.

The next meeting of the society will be held jointly with the Indiana Academy of Ophthalmology and Otolaryngology at French Lick, Indiana, April 24th, 25th, and 26th.

DALLAS AND FORT WORTH

Meetings of the Dallas Academy of Ophthalmology and Otolaryngology and Fort Worth EENT Society have been held on September 5, October 7,

November 4, and December 2, 1958, and January 6, 1959. Meetings for the remainder of the year are on Friday, February 6th, when Dr. Miles L. Lewis, Jr., Ochsner Clinic, New Orleans, will be the guest speaker; Friday, February 20th, when Dr. Charles L. Schepens, Boston, will be the guest speaker; Tuesday, April 7th, Dr. Samuel L. Fox, Baltimore, guest speaker.

During March the Dallas Southern Clinical Society will hold its annual meeting at which the guest speakers for Ophthalmology will be Dr. Frank Costenbader, Washington, D.C., Dr. Irving H. Leopold, Philadelphia, Dr. Frank Lathrop, Boston, and Dr. Howard House, Los Angeles. The social meeting on May 1st will be held at the Cipango Club, and at the June 2nd meeting, Dr. Carrol Browning, Dallas, will discuss "The application of electronic microscope and ultrasonic techniques to the pathology of the eye."

Officers of the societies are: Dallas—President, Dr. Edward A. Newell; vice-president, Dr. Thomas M. McCrory; secretary-treasurer, Dr. James L. Baldwin; program co-chairmen, Dr. Ronald M. Burnside and Dr. Daniel M. Martinez. Fort Worth—President, Dr. Van D. Rathgeber; vice-president, Dr. William Skokan; secretary-treasurer, Dr. Paul Rockwell.

Oxford Ophthalmological Congress

The next meeting of the Oxford Ophthalmological Congress will be held on July 1st, 2nd, 3rd, and 4th, at which time the society will celebrate the 50th anniversary of its founding. Recently elected officers are: Master, Mr. O. M. Duthie, Manchester; deputy master, Mr. C. H. Bamford, Derby; editorial secretary, Mr. L. P. Jameson Evans, Birmingham; and hon. secretary and treasurer, Mr. Ian C. Fraser, Shrewsbury.

The main topic for discussion at the 1959 meeting will be "Ophthalmic manifestations in pediatric practice." One day will be devoted to "After thoughts" on the Doyne Lecture by 15 past lecturers.

Oklahoma City meeting

The annual spring meeting of the Oklahoma City Academy of Ophthalmology and Otolaryngology will be held March 12th and 13th. The guest lecturers in ophthalmology are Dr. Michael J. Hogan, San Francisco, and Dr. Ralph O. Rychener, Memphis. The guest speaker in otolaryngology will be Dr. Ramon del Villar, Mexico City. For further details communicate with Dr. E. Norris Robertson, Jr., 301 N.W. 12th Street, Oklahoma City, Oklahoma.

VI Pan-American congress of ophthalmology

The VI Pan-American Congress of Ophthalmology will convene in Caracas, Venezuela, from January 31 to February 7, 1960.

The scientific program includes symposia on virus diseases in ophthalmology, toxoplasmosis, genetics, strabismus, detachment of the retina, medical advances in ophthalmology, surgical advances in ophthalmology, therapy of infectious diseases, surgery of strabismus, surgery of cataract, and space ophthalmology.

Free papers will be accepted and should be submitted to the chairman of the Program Committee, Dr. James H. Allen (1430 Tulane Avenue, New Orleans, Louisiana, U.S.A.) to June 1, 1959.

Movies will also be accepted provided they have not been shown previously and that the author will be present at the meeting to answer any questions regarding the movie.

There will be a scientific exhibition. Details about this exhibition can be obtained from Dr. James H. Allen.

The congress has the full backing of the government of Venezuela and the government of that country especially wants to welcome all ophthalmologists from the Americas. Caracas is a very beautiful city with an altitude of 2,700 feet which makes its climate quite temperate, especially in January and February. It has very good hotels and is linked by a superhighway with the port of La Guaira where the airport for Caracas is also located. The trip from La Guaira to Caracas takes only 20 minutes.

It has been decided that reservations should be made directly to Mr. Gus A. Romea, Hotel Tamanaco, Caracas, Venezuela, who will assign rooms to those who request them, according to the dates of the requests. The Tamanaco Hotel will be the headquarters of the congress. The scientific meetings will be held at the university where there is a very large auditorium equipped with earphones, thus facilitating simultaneous translation.

Prices in Caracas are no higher than they are in the United States and there is a very large number of United States citizens living in Venezuela, around 25,000.

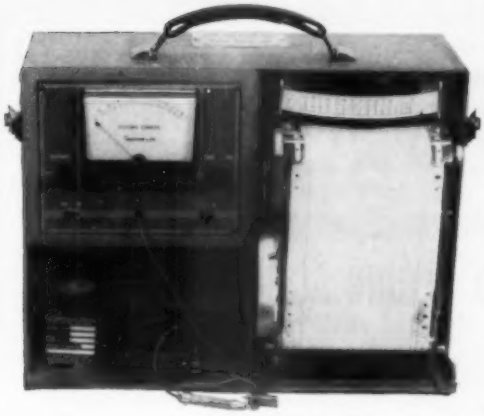
The local committee has planned a very interesting social program, so that the VI Pan-American Congress of Ophthalmology will be as good as the others which preceded it.

Please write to Dr. Moacyr E. Alvaro, executive director of the Pan-American Association of Ophthalmology, 1151 Consolacão, São Paulo, Brazil, for any further information regarding the meeting. You can also write to Dr. J. Wesley McKinney, our secretary for north of Panama (Suite 921, Exchange Building, Memphis, Tennessee, U.S.A.) and to Dr. William L. Benedict (15 Second Street, S. W., Rochester, Minnesota, U.S.A.), the chairman of the Committee on Congresses.

Personals

Among the newcomers elected to the 86th Congress is Dr. T. Dale Alford, of Little Rock, Arkansas. Dr. Alford received his certificate from the American Board of Ophthalmology in 1948. He was graduated (M.D.) from the University of Arkansas and was a professor of ophthalmology at the University of Arkansas prior to his World War II service in the U. S. Army Medical Corps.

The American Journal of Ophthalmology salutes him and extends all good wishes.



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